

CLIENT'S NAME AND ADDRESS:
ACROFEMI HEALTHCARE LTD (MEDIWHEEL)
F-703, F-703, LADO SARAI, MEHRAULI

DUVCTON EVANINATION LIDINE

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

PATIENT ID: RESHF100172181

Email: customercare.thane@srl.in

PATIENT NAME: RESHMA GULRAJANI

ACCESSION NO: 0181VI000320 AGE: 50 Years SEX: Female

DRAWN: RECEIVED: 10/09/2022 10:02 REPORTED: 13/09/2022 15:18

REFERRING DOCTOR: SELF CLIENT PATIENT ID:

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

MEDI WHEEL FULL BODY HEALTH CHECKUP ABOVE 40FEMALE

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	12.0		12.0 - 15.0	g/dL
METHOD : SLS- HEMOGLOBIN DETECTION METHOD				
RED BLOOD CELL COUNT	4.45		3.8 - 4.8	mil/µL
METHOD: HYDRODYNAMIC FOCUSING BY DC DETECTION				
WHITE BLOOD CELL COUNT	5.82		4.0 - 10.0	thou/µL
METHOD: FLUORESCENCE FLOW CYTOMETRY				
PLATELET COUNT	268		150 - 410	thou/µL
METHOD: HYDRODYNAMIC FOCUSING BY DC DETECTION				
RBC AND PLATELET INDICES				
HEMATOCRIT	37.5		36.0 - 46.0	%
METHOD: CUMULATIVE PULSE HEIGHT DETECTION METHOD				
MEAN CORPUSCULAR VOL	84.3		83.0 - 101.0	†L
METHOD: CALCULATED FROM RBC & HCT				
MEAN CORPUSCULAR HGB.	27.0		27.0 - 32.0	pg
METHOD: CALCULATED FROM THE RBC & HGB				
MEAN CORPUSCULAR HEMOGLOBIN	32.0		31.5 - 34.5	g/dL
CONCENTRATION METHOD: CALCULATED FROM THE HGB & HCT				
MENTZER INDEX	18.9			
RED CELL DISTRIBUTION WIDTH	13.6		11.6 - 14.0	%
METHOD: CALCULATED FROM RBC SIZE DISTRIBUTION CURVE				
MEAN PLATELET VOLUME	11.3	High	6.8 - 10.9	fL
METHOD: CALCULATED FROM PLATELET COUNT & PLATELET HEMATO	OCRIT			
CHEMICAL EXAMINATION, URINE				
PH	7.0		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			
UROBILINOGEN	NORMAL	NORMAL	
METHOD: MODIFIED EHRLICH REACTION			
NITRITE	NOT DETECTED	NOT DETECTED	
METHOD: 1,2,3,4-TETRAHYDROBENZO(H)QUINOLIN-3-OL	NOT DETENTED	NOT DETENTED	
LEUKOCYTE ESTERASE	NOT DETECTED	NOT DETECTED	
WBC DIFFERENTIAL COUNT - NLR			
SEGMENTED NEUTROPHILS	58	40 - 80	%
METHOD: FLOW CYTOMETRY WITH LIGHT SCATTERING			
ABSOLUTE NEUTROPHIL COUNT	3.40	2.0 - 7.0	thou/µL
METHOD: FLOW CYTOMETRY WITH LIGHT SCATTERING			
LYMPHOCYTES	34	20 - 40	%
METHOD: FLOW CYTOMETRY WITH LIGHT SCATTERING	4.05	4.0.00	
ABSOLUTE LYMPHOCYTE COUNT	1.95	1.0 - 3.0	thou/µL
METHOD: FLOW CYTOMETRY WITH LIGHT SCATTERING	4 7		
NEUTROPHIL LYMPHOCYTE RATIC (NLR)	1.7		0/
EOSINOPHILS	3	1 - 6	%
METHOD: FLOW CYTOMETRY WITH LIGHT SCATTERING	0.47	0.00 0.50	41 <i>f</i> 1
ABSOLUTE EOSINOPHIL COUNT	0.17	0.02 - 0.50	thou/µL
METHOD: FLOW CYTOMETRY WITH LIGHT SCATTERING	5	2 - 10	%
MONOCYTES METHOD: FLOW CYTOMETRY WITH LIGHT SCATTERING	J	2 - 10	70
ABSOLUTE MONOCYTE COUNT	0.26	0.2 - 1.0	thou/µL
METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING	0.20	0.2 - 1.0	поальс
DIFFERENTIAL COUNT PERFORMED ON:	EDTA SMEAR		
MICROSCOPIC EXAMINATION, URINE	ED LA OMENIA		
	5-7	0-5	/HPF
PUS CELL (WBC'S) METHOD: MICROSCOPIC EXAMINATION	J-1	0-3	/ FIRE
EPITHELIAL CELLS	1-2	0-5	/HPF
METHOD: MICROSCOPIC EXAMINATION	1 4	<u> </u>	/ L IEI
ERYTHROCYTES (RBC'S)	NOT DETECTED	NOT DETECTED	/HPF
METHOD: MICROSCOPIC EXAMINATION	.,0, 52,20,25		,
CASTS	NOT DETECTED		
	· - · · - - · · ·		







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METHOD: MICROSCOPIC EXAMINATION	NOT DETENTED			
CRYSTALS	NOT DETECTED			
METHOD: MICROSCOPIC EXAMINATION	NOT DETECTED		NOT DETECTED	
BACTERIA METHOD: MICROSCOPIC EXAMINATION	NOT DETECTED		NOT DETECTED	
YEAST	NOT DETECTED		NOT DETECTED	
	NOT DETECTED		NOT DETECTED	
MORPHOLOGY	NODMOOVETO NODM	201.120	NATO.	
RBC	NORMOCYTIC NORMO		DMIC	
WBC	NORMAL MORPHOLOG	GΥ		
METHOD: MICROSCOPIC EXAMINATION	AREQUATE			
PLATELETS	ADEQUATE			
ERYTHRO SEDIMENTATION RATE, BLOOD				
SEDIMENTATION RAIL (ESR)	18		0 - 20	mm at 1 hr
METHOD: WESTERGREN METHOD				
GLYCOSYLATED HEMOGLOBIN, EDTA WHOLE B				
GLYCOSYLATED HEMOGLOBIN (HBA1C)	6.2	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	131.2	Hiab	< 116.0	ma/dL
METHOD : CALCULATED PARAMETER	131.2	mgn	< 110.0	mg/aL
GLUCOSE, FASTING, PLASMA				
GLUCOSE, FASTING, PLASMA	98		Normal 75 - 99 Pre-diabetics: 100 - 125 Diabetic: > or = 126	mg/dL
METHOD: ENZYMATIC REFERENCE METHOD WITH HEXOKINASE			Diabetic. > 0 = 120	
GLUCOSE, POST-PRANDIAL, PLASMA				
GLUCOSE, POST-PRANDIAL, PLASMA	105		70 - 139	mg/dL
METHOD: ENZYMATIC REFERENCE METHOD WITH HEXOKINASE				,
CORONARY RISK PROFILE, SERUM				
CHOLESTEROL	223	High	Desirable cholesterol level < 200 Borderline high cholesterol 200 - 239 High cholesterol > / = 240	mg/dL
METHOD: ENZYMATIC COLORIMETRIC ASSAY				

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TRIGLYCERIDES METHOD: ENZYMATIC COLORIMETRIC ASSAY	84		Normal: < 150 Borderline high: 150 - 199 High: 200 - 499 Very High: >/= 500	mg/dL
HDL CHOLESTEROL	48.3		Low HDL Cholesterol <40	mg/dL
METHOD : ENZYMATIC, COLORIMETRIC	1010		High HDL Cholesterol >/= 60	-
CHOLESTEROL LDL	158	High	Adult levels: Optimal < 100 Near optimal/above optimal: 1 129 Borderline high: 130-159 High: 160-189 Very high: = 190	mg/dL 00-
METHOD: ENZYMATIC COLORIMETRIC ASSAY			, ,	
NON HDL CHOLESTEROL	175	High	Desirable : < 130 Above Desirable : 130 -159 Borderline High : 160 - 189 High : 190 - 219 Very high : > / = 220	mg/dL
CHOL/HDL RATIO	4.6	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	3.3	High	0.5 - 3.0 Desirable/Low Risk 3.1 - 6.0 Borderline/Moderate >6.0 High Risk	Risk
VERY LOW DENSITY LIPOPROTEIN	16.8		< OR = 30.0	mg/dL
LIVER FUNCTION PROFILE, SERUM				
BILIRUBIN, TOTAL METHOD: COLORIMETRIC DIAZO	0.23		Upto 1.2	mg/dL
BILIRUBIN, DIRECT	0.13		< 0.30	mg/dL
BILIRUBIN, INDIRECT	0.10		0.1 - 1.0	mg/dL
TOTAL PROTEIN METHOD: COLORIMETRIC	7.3		6.0 - 8.0	g/dL
ALBUMIN METHOD: COLORIMETRIC	4.8		3.97 - 4.94	g/dL
GLOBULIN	2.5		2.0 - 3.5	g/dL
ALBUMIN/GLOBULIN RATIO	1.9		1.0 - 2.1	RATIO
ASPARTATE AMINOTRANSFERASE (AST/SGOT) METHOD: UV ABSORBANCE	21		< OR = 35	U/L







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ALANINE AMINOTRANSFERASE (ALT/SGPT)	14		< OR = 35	U/L
METHOD: UV ABSORBANCE	125	⊔iah	35 - 104	1.171
ALKALINE PHOSPHATASE METHOD: COLORIMETRIC	123	nigii	33 - 104	U/L
GAMMA GLUTAMYL TRANSFERASE (GGT)	13		0 - 40	U/L
METHOD: ENZYMATIC, COLORIMETRIC	10		0 10	٥, ۵
LACTATE DEHYDROGENASE	166		125 - 220	U/L
METHOD: UV ABSORBANCE				
SERUM BLOOD UREA NITROGEN				
BLOOD UREA NITROGEN	11		6 - 20	mg/dL
METHOD: ENZYMATIC ASSAY				
CREATININE, SERUM				
CREATININE	0.67		0.5 - 0.9	mg/dL
METHOD: COLORIMETRIC				
BUN/CREAT RATIO				
BUN/CREAT RATIO	16.42	High	8.0 - 15.0	
URIC ACID, SERUM				
URIC ACID	6.5	High	2.4 - 5.7	mg/dL
METHOD: ENZYMATIC COLORIMETRIC ASSAY				
TOTAL PROTEIN, SERUM				
TOTAL PROTEIN	7.3		6.0 - 8.0	g/dL
METHOD: COLORIMETRIC				
ALBUMIN, SERUM				
ALBUMIN	4.8		3.97 - 4.94	g/dL
METHOD: COLORIMETRIC				
GLOBULIN				
GLOBULIN	2.5		2.0 - 3.5	g/dL
ELECTROLYTES (NA/K/CL), SERUM				
SODIUM	144		136 - 145	mmol/L
POTASSIUM	4.50		3.5 - 5.1	mmol/L
CHLORIDE	103		98 - 107	mmol/L
THYROID PANEL, SERUM				
Т3	88.3		80 - 200	ng/dL
METHOD: ELECTROCHEMILUMINESCENCE				
T4	6.38		5.1 - 14.1	μg/dL
METHOD: ELECTROCHEMILUMINESCENCE				
TSH 3RD GENERATION	1.270		0.27 - 4.2	μIU/mL







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METHOD: ELECTROCHEMILUMINESCENCE

PAPANICOLAOU SMEAR

TEST METHOD CONVENTIONAL GYNEC CYTOLOGY

SPECIMEN TYPE P 1017/22

TWO UNSTAINED CERVICAL SMEARS RECEIVED

2014 BETHESDA SYSTEM FOR REPORTING CERVICAL CYTOLOGY REPORTING SYSTEM

SPECIMEN ADEQUACY SATISFACTORY

THE SMEARS SHOW MAINLY SUPERFICIAL SQUAMOUS CELLS, FEW MICROSCOPY

INTERMEDIATE SQUAMOUS CELLS, AND FEW CLUSTERS OF ENDOCERVICAL CELLS IN THE BACKGROUND OF MODERATE

POLYMORPHS

INTERPRETATION / RESULT NEGATIVE FOR INTRAEPITHELIAL LESION OR MALIGNANCY

ENDOMETRIAL CELLS (IN A WOMAN >/= 45 YRS) ABSENT

Comments

PLEASE NOTE PAPANICOLAU 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 B

METHOD: GEL COLUMN AGGLUTINATION METHOD.

RH TYPE **POSITIVE**

METHOD: GEL COLUMN AGGLUTINATION METHOD.

XRAY-CHEST

NO ABNORMALITY DETECTED **IMPRESSION**

TMT OR ECHO

TMT OR ECHO 2D ECHO :-

Structurally normal valves. Mild TR

Concentric LVH.

No regional wall motion abnormality.

Good Left Ventricular systolic function, LVEF 60 %

Normal LV Diastolic function. No e/o pulmonary hypertension.

ECG

ECG WITHIN NORMAL LIMITS

MAMOGRAPHY (BOTH BREASTS)

MAMOGRAPHY 30TH BREASTS NORMAL

MEDICAL HISTORY



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RELEVANT PRESENT HISTORY HYPERTENSIV SINCE 8 YEARS.

RELEVANT PAST HISTORY L3-L4 SLIP DISC TAKEN PHYSIOTHERAPY. COVID IN JAN 2022. HOME QUARANTINED.

RELEVANT PERSONAL HISTORY MARRIED / 2 CHILD / MIXED DIET / NO ALLERGIES / NO SMOKING /

OCC ALCÓHOL. MENOPAUSAL 2FTNDA0L2.

OBSTETRIC HISTORY (FOR FEMALES) 24 YEARS BACK. LCB (FOR FEMALES) RELEVANT FAMILY HISTORY DIABETES: FATHER. HISTORY OF MEDICATIONS OLMESAR 10: 1-0-0.

ANTHROPOMETRIC DATA & BMI

MENSTRUAL HISTORY (FOR FEMALES)

HEIGHT IN METERS 1.55 mts WEIGHT IN KGS. 80 Kgs

RMI BMI & Weight Status as follows: kg/sqmts 33 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 OBESE 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 86/MIN.REGULAR, ALL PERIPHERAL PULSES WELL FELT, NO CAROTID

BRUIT

RESPIRATORY RATE NORMAL

CARDIOVASCULAR SYSTEM

ΒP 180/100 MM HG mm/Hg

(SUPINE)







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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		
EYE MOVEMENTS	NORMAL		
CORNEA	NORMAL		
DISTANT VISION RIGHT EYE WITHOUT GLASSES	REDUCED VISUAL ACUITY	6/9	
DISTANT VISION LEFT EYE WITHOUT GLASSES	REDUCED VISUAL ACUITY	6/12	

WITHIN NORMAL LIMIT



NEAR VISION RIGHT EYE WITHOUT GLASSES





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NEAR VISION LEFT EYE WITHOUT GLASSES WITHIN NORMAL LIMIT

COLOUR VISION NORMAL

SUMMARY

RELEVANT HISTORY NOT SIGNIFICANT RELEVANT GP EXAMINATION FINDINGS OBESE: BMI 33

REDUCED VISUAL ACUITY FOR DISTANT VISION.

UROLOGY PHYSICIAN CONSULT FOR CONTROL OF BP.

STRICT LOW FAT, LOW CALORIE, LOW CARBOHYDRATE, HIGH FIBRE

DIET.

3) REGULAR EXERCISE REGULAR WALK FOR 30-40 MIN DAILY.

4) REPEAT B.SUGAR, LIPID PROFILE AFTER 3 MONTHS OF DIET AND

EXERCISE.

OPHTHALMOLOGY CONSULT FOR REDUCED VISUAL ACUITY.

6) SURGICAL CONSULT SOS FOR UMBILICAL HERNIA.

Interpretation(s)

REMARKS / RECOMMENDATIONS

BLOOD COUNTS, EDTA WHOLE BLOODThe 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

Ketones: Uncontrolled diabetes mellitus car lead to presence of ketones in urine, Ketones can also be seen in starvation, frequent vomiting, pregnancy and strenuous exercise.

Blood: Occult blood can occur in urine as intact erythrocytes or haemoglobin, which can occur in various urological, nephrological and bleeding disorders. Leukocytes: An increase in leukocytes is an indication of inflammation in urinary tract or kidneys. Most common cause is bacterial urinary tract infection.

Nitrite: Many bacteria give positive results when their number is high. Nitrite concentration during infection increases with length of time the urine specimen is retained in bladder prior to collection.
pH: The kidneys play an important role in maintaining 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 concentratec the urine is. Increasec specific gravity is seen in conditions like dehydration, glycosuria and

proteinuria while decreased specific gravity is seen in excessive fluid intake, renal failure and diabetes insipidus.

Bilirubin: In certain liver diseases such as biliary obstruction or hepatitis, bilirubin gets excreted in urine. Urobilinogen: Positive results are seen in liver diseases like hepatitis and cirrhosis and in cases of hemolytic anemia

ERYTHRO SEDIMENTATION RATE, BLOODErythrocyte 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:

Nathan and Oski's Haematology of Infancy and Childhood, 5th edition
 Paediatric reference intervals. AACC Press, 7th edition. Edited by S. Soldin
 The reference for the adult reference range is "Practical Haematology by Dacie and Lewis, 10th Edition".

GLYCOSYLATED HEMOGLOBIN, EDTA WHOLE BLOOD-







ACROFEMI HEALTHCARE LTD (MEDIWHEEL) 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: RESHMA GULRAJANI

PATIENT ID: RESHF100172181

ACCESSION NO: 0181VI000320 AGE: 50 Years SEX: Female

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Glycosylatec 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 reciblood 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 afters 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 seventy 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 rec cells.

Glycosylated hemoglobins results from patients with HbSC, and HbSC and HbSC

increased rec cell turnover, transfusion requirements, that adversely impact HbA1c as a marker of long-term glycemic control. Ir these conditions, alternative forms of testing such as glycated serum protein (fructosamine) should be considered.

Targets should be individualized; More or less stringent glycemic goals may be appropriate for individual patients. Goals should be individualized based on duration of diabetes, age/life expectancy, comorbid conditions, known CVD or advanced microvascular complications, hypoglycemia unawareness, and individual patient considerations.

References

- 1. Tietz Textbook of Clinical Chemistry and Molecular Diagnostics, edited by Carl A Burtis, Edward R.Ashwood, David E Bruns, 4th Edition, Elsevier publication, 2006, 879-884.
- 2. Forsham PH. Diabetes Mellitus: A rational plan for management. Postgrad Med 1982, 71,139-154.
- 3. Mayer TK, Freedman ZR: Protein glycosylation in Diabetes Mellitus: A review of laboratory measurements and their clinical utility. Clin Chim Acta 1983, 127, 147-184. GLUCOSE, FASTING, PLASMA-

ADA 2021 guidelines for adults, after 8 hrs fasting is as follows: Pre-diabetics: 100 - 125 mg/dL

Diabetic: > or = 126 mg/dL

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

LIVER FUNCTION PROFILE, SERUM-LIVER FUNCTION PROFILE

Bilirubin is a yellowish pigment founc in bile and is a breakdowr product of normal heme catabolism. Bilirubin is excreted in bile and urine, and elevated levels may give yellow discoloration in jaundice. Elevatec levels results from increased bilirubir 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 termec 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, hemotytic 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 bound mainly in the liver, but also in smaller amounts in the kidneys, heart, muscles, and pancreas. It is commonly measured as a part of a diagnostic evaluation of hepatocellular injury, to determine liver health. AST levels increase during acute hepatitis, sometimes due to a viral infection, ischemia to the liver, chronic hepatitis, obstruction of bile ducts, cirrhosis.

ALP is a protein found in almost all body tissues. Tissues with higher amounts of ALP include the liver, bile ducts and bone. Elevated ALP levels are seen in Biliary obstruction, Osteoblastic bone tumors, osteomalacia, hepatitis, Hyperparathyroidism, Leukemia, Lymphoma, Paget's disease, Rickets, Sarcoidosis etc. Lower-than-normal ALP levels seen in Hypophosphatasia, Malnutrition, Protein deficiency, Wilson's disease. GGT is an enzyme found in cell membranes of many tissues mainly in the liver, kidney and pancreas. It is also found in other tissues including intestine, spleen, heart, brain and seminal vesicles. The highest concentration is in the kidney, but the liver is considered the source of normal enzyme activity. Serum GGT has been widely used as an index of liver dystunction. 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 consumptor 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 albumir 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, mainutrition and wasting etc

SERUM BLOOD UREA NITROGEN-

Causes of Increased levels

Pre renal

- High protein diet, Increasec protein catabolism, GI haemorrhage, Cortisol, Dehydration, CHF Renal
- Renal Failure
- 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



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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

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• Loss of body fluid (dehydration)

· Muscle problems, such as breakdown of muscle fibers

· Problem's 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

Multiple Scierosis

Nutritional tips to manage increased Uric acic levels
• Drink plenty of fluids

· Limit animal proteins

High Fibre foods

Vit C Intake

Antioxidant rich foods TOTAL PROTEIN, SERUM-

Serum total protein, also known as total protein, is a biochemical test for measuring the total amount of protein in serum. Protein in the plasma is made up of albumin and

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 albumir is the most abundant protein in human blooc plasma. It is produced in the liver. Albumin constitutes about half of the blooc serum protein. Low blood albumin levels (hypoalbuminemia) can be 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), SERUMSodium 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, Addisoniar crisis, certain types of metabolic acidosis, persistent gastric secretion and

respiratory actions, Sale-tusing reprintus, inecapone area of a serious and serious and serious and processes of the process o

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

TOTAL TÃ TOTAL T4 Levels in (µg/dL) 6.6 **-** 12.4 (ng/dL) 81 - 190 Pregnancy (µIU/mL) 0.1 - 2.5 First Trimester

2nd Trimester 6.6 - 15.5 6.6 - 15.5 0.2 - 3.0 0.3 - 3.0 100 - 260 100 - 260 3rc Trimester Below mentioned are the guidelines for age related reference ranges for T3 and T4.

T3 (µg/dL) 1-3 day: 8.2 - 19.9 (ng/dL) New Born: 75 - 260 1 Week: 6.0 - 15.9



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SOUTH WEST DELHI NEW DELHI 110030 DELHI INDIA 8800465156

SRL Ltd S.K. Tower, Hari Niwas, LBS Marg THANE, 400602

MAHARASHTRA, INDIA

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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 or 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.

Gowenlock A.H. Varley's Practical Clinical Brochemistry, 6th Edition.
 Behrman R.E. Kilegman R.M., Jenson H. B. Nelsor 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: "Flease 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.

THIS REPORT CARRIES THE SIGNATURE OF OUR LABORATORY DIRECTOR, THIS IS AN INVIOLABLE FEATURE OF OUR LAB MANAGEMENT SOFTWARE, HOWEVER, ALL EXAMINATIONS AND INVESTIGATIONS HAVE BEEN CONDUCTED BY OUR PANEL OF DOCTORS.







CLIENT'S NAME AND ADDRESS:
ACROFEMI HEALTHCARE LTD (MEDIWHEEL)
F-703, F-703, LADO SARAI, MEHRAULI

SOUTH WEST DELHI NEW DELHI 110030 DELHI INDIA 8800465156

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MEDI WHEEL FULL BODY HEALTH CHECKUP ABOVE 40FEMALE

ULTRASOUND ABDOMEN ULTRASOUND ABDOMEN GRADE | FATTY LIVER TINY UMBILICAL HERNIA.

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

Dr.Priyal Chinchkhede

Bhinchkhede

Consultant Pathologist

Dr. Ushma Wartikar Consultant Pathologist

Dr.(Mrs)Neelu K Bhojani

Lab Head



