

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

PATIENT ID:

SUPRF030479L

Email: customercare.thane@srl.in

PATIENT NAME: SUPRIYA DIWAKAR

ACCESSION NO: 0181VI000303 AGE: 43 Years SEX: Female

DRAWN: RECEIVED: 10/09/2022 09:14 REPORTED: 13/09/2022 15:20

REFERRING DOCTOR: SELF CLIENT PATIENT ID:

Test Report Status <u>Final</u> Results Biological Reference Interval Units

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	10.7	Low 12.0 - 15.0	g/dL
METHOD: SLS-HEMOGLOBIN DETECTION METHOD			
RED BLOOD CELL COUNT	4.48	3.8 - 4.8	mil/µL
METHOD: HYDRODYNAMIC FOCUSING BY DC DETECTION			

WHITE BLOOD CELL COUNT 7.13 4.0 - 10.0

METHOD: FLUORESCENCE FLOW CYTOMETRY

PLAIELEI COUNT 101 Low 150 - 410 thou/µL

METHOD: HYDRODYNAMIC FOCUSING BY DC DETECTION

RBC AND PLATELET INDICES

HEMATOCRIT	33.9	Low 36.0 - 46.0	%
METHOD: CUMULATIVE PULSE HEIGHT DETECTION METHOD			
MEAN CORDUSCULAR VOL	75.7	Low 83 N - 101 N	Ħ

METHOD : CALCULATED FROM RBC & HCT

MEAN CORPUSCULAR HGB.

23.9

Low 27.0 - 32.0 pg

METHOD : CALCULATED FROM THE RBC & HGB

MEAN CORPUSCULAR HEMOGLOBIN 31.6 31.5 - 34.5 g/dL

CONCENTRATION

METHOD: CALCULATED FROM THE HGB & HCT

MENTZER INDEX 16.9

RED CELL DISTRIBUTION WIDTH 18.3 High 11.6 - 14.0 %

 ${\tt METHOD: CALCULATED\ FROM\ RBC\ SIZE\ DISTRIBUTION\ CURVE}$

CHEMICAL EXAMINATION, URINE

P- 6.0 4.7 - 7.5

METHOD: DOUBLE INDICATOR PRINCIPLE

PROTEIN NOT DETECTED NOT DETECTED

METHOD: TETRA BROMOPHENOL BLUE/SULFOSALICYLIC ACID

GLUCOSE NOT DETECTED NOT DETECTED

METHOD: GLUCOSE OXIDASE PEROXIDASE





thou/µL



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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	60	40 - 80	%
METHOD: FLOW CYTOMETRY WITH LIGHT SCATTERING			
ABSOLUTE NEUTROPHIL COUNT	4.28	2.0 - 7.0	thou/µL
METHOD: FLOW CYTOMETRY WITH LIGHT SCATTERING			
LYMPHOCYTES	35	20 - 40	%
METHOD: FLOW CYTOMETRY WITH LIGHT SCATTERING			
ABSOLUTE LYMPHOCYTE COUNT	2.50	1.0 - 3.0	thou/µL
METHOD: FLOW CYTOMETRY WITH LIGHT SCATTERING			
NEUTROPHIL LYMPHOCYTE RATIC (NLR)	1.7		
EOSINOPHILS	02	1 - 6	%
METHOD: FLOW CYTOMETRY WITH LIGHT SCATTERING			
ABSOLUTE EOSINOPHIL COUNT	0.14	0.02 - 0.50	thou/µL
METHOD: FLOW CYTOMETRY WITH LIGHT SCATTERING			
MONOCYTES	03	2 - 10	%
METHOD: FLOW CYTOMETRY WITH LIGHT SCATTERING			
ABSOLUTE MONOCYTE COUNT	0.21	0.2 - 1.0	thou/µL
METHOD: FLOW CYTOMETRY WITH LIGHT SCATTERING	EDT A OMEAD		
DIFFERENTIAL COUNT PERFORMED ON:	EDTA SMEAR		
MICROSCOPIC EXAMINATION, URINE			
PUS CELL (WBC'S)	3-5	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	NOT DETENTED		
CRYSTALS	NOT DETECTED		







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METHOD: MICROSCOPIC EXA	MOITANIM				
BACTERIA		NOT DETECTED		NOT DETECTED	
METHOD: MICROSCOPIC EXA	MINATION				
YEAST		NOT DETECTED		NOT DETECTED	
MORPHOLOGY					
RBC		HYPOCHROMASIA & M	MICRO	CYTOSSIS	
WBC		REACTIVE LYMPHOCY	YTES S	EEN	
METHOD: MICROSCOPIC EXA	MINATION				
PLATELETS				ΓON SMEAR APPEARS TO BE 10	0-110
EDVELIDO GEDINAENE	TION DATE DI COD	THOU/uL,LARGE PLAT	TELETS	SSEEN	
ERYTHRO SEDIMENTA	•				
SEDIMENTATION RATE (` ′	35	High	0 - 20	mm at 1 hr
METHOD : WESTERGREN MET					
	OGLOBIN, EDTA WHOLE BI				
GLYCOSYLATED HEMOG	LOBIN (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 GLUCOSI		105.4		< 116.0	mg/dL
METHOD : CALCULATED PARA					
GLUCOSE, FASTING, F					
GLUCOSE, FASTING, PLA	ASMA	95		Normal 75 - 99 Pre-diabetics: 100 - 125 Diabetic: > or = 126	mg/dL
METHOD: ENZYMATIC REFER	ENCE METHOD WITH HEXOKINASE				
GLUCOSE, POST-PRAN	NDIAL, PLASMA				
GLUCOSE, POST-PRAND	IAL, PLASMA	107		70 - 139	ma/dL

GLUCOSE, POST-PRANDIAL, PLASMA 107 70 - 139 mg/dL METHOD: ENZYMATIC REFERENCE METHOD WITH HEXOKINASE

CORONARY RISK PROFILE, SERUM

CHOLESTEROL 144 Desirable cholesterol level

< 200

Borderline high cholesterol

200 - 239 High cholesterol > / = 240

METHOD: ENZYMATIC COLORIMETRIC ASSAY





mg/dL



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TRIGLYCERIDES METHOD: ENZYMATIC COLORIMETRIC ASSAY	65	Normal: < 150 mg/dL Borderline high: 150 - 199 High: 200 - 499 Very High: >/= 500	
HDL CHOLESTEROL	49.7	Low HDL Cholesterol <40 mg/dL	
METHOD : ENZYMATIC, COLORIMETRIC	1311	High HDL Cholesterol >/= 60	
CHOLESTEROL LDL	81	Adult levels: mg/dL Optimal < 100 Near optimal/above optimal: 100- 129 Borderline high : 130-159 High : 160-189 Very high : = 190	
METHOD: ENZYMATIC COLORIMETRIC ASSAY		,g 250	
NON HDL CHOLESTEROL	94	Desirable : < 130 mg/dL Above Desirable : 130 -159 Borderline High : 160 - 189 High : 190 - 219 Very high : > / = 220	
CHOL/HDL RATIO LDL/HDL RATIO	2.9 1.6	Low Low Risk: 3.3 - 4.4 Average Risk: 4.5 - 7.0 Moderate Risk: 7.1 - 11.0 High Risk: > 11.0 0.5 - 3.0 Desirable/Low Risk	
		3.1 - 6.0 Borderline/Moderate Risk >6.0 High Risk	
VERY LOW DENSITY LIPOPROTEIN	13.0	< OR = 30.0 mg/dL	
LIVER FUNCTION PROFILE, SERUM			
BILIRUBIN, TOTAL METHOD : COLORIMETRIC DIAZO	0.59	Upto 1.2 mg/dL	
BILIRUBIN, DIRECT	0.30	< 0.30 mg/dL	
BILIRUBIN, INDIRECT	0.29	0.1 - 1.0 mg/dL	
TOTAL PROTEIN METHOD: COLORIMETRIC	8.3	High 6.0 - 8.0 g/dL	
ALBUMIN METHOD: COLORIMETRIC	4.5	3.97 - 4.94 g/dL	
GLOBULIN	3.8	High 2.0 - 3.5 g/dL	
ALBUMIN/GLOBULIN RATIO	1.2	1.0 - 2.1 RATIO	
ASPARTATE AMINOTRANSFERASE (AST/SGOT) METHOD: UV ABSORBANCE	21	< OR = 35 U/L	







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AL ANITALE ANATALOTO AND	EEDAGE (ALT/OCCT)	10			
ALANINE AMINOTRANS METHOD: UV ABSORBANCE	* '	18		< OR = 35	U/L
ALKALINE PHOSPHATAS		55		35 - 104	U/L
METHOD : COLORIMETRIC	JL .	33		33 104	O/ L
GAMMA GLUTAMYL TRA	NSFERASE (GGT)	5		0 - 40	U/L
METHOD : ENZYMATIC, COLO	ORIMETRIC				
LACTATE DEHYDROGEN	NASE	140		125 - 220	U/L
METHOD: UV ABSORBANCE					
SERUM BLOOD UREA	NITROGEN				
BLOOD UREA NITROGE	:N	6		6 - 20	mg/dL
METHOD: ENZYMATIC ASSA					
CREATININE, SERUM	1				
CREATININE		0.60		0.5 - 0.9	mg/dL
METHOD : COLORIMETRIC					
BUN/CREAT RATIO					
BUN/CREAT RATIO		10.00		8.0 - 15.0	
URIC ACID, SERUM					
URIC ACID		4.8		2.4 - 5.7	mg/dL
METHOD : ENZYMATIC COLO	DRIMETRIC ASSAY				
TOTAL PROTEIN, SER	RUM				
TOTAL PROTEIN		8.3	High	6.0 - 8.0	g/dL
METHOD: COLORIMETRIC					
ALBUMIN, SERUM					
ALBUMIN		4.5		3.97 - 4.94	g/dL
METHOD: COLORIMETRIC					
GLOBULIN					
GLOBULIN		3.8	High	2.0 - 3.5	g/dL
ELECTROLYTES (NA/	K/CL), SERUM				
SODIUM		139		136 - 145	mmol/L
POTASSIUM		4.77		3.5 - 5.1	mmol/L
CHLORIDE		102		98 - 107	mmol/L
THYROID PANEL, SER	RUM				
Т3		107.0		80 - 200	ng/dL
METHOD : ELECTROCHEMILL	JMINESCENCE				<u>-</u> -
T4		9.45		5.1 - 14.1	µg/dL
METHOD : ELECTROCHEMILL	JMINESCENCE				
TSH 3RD GENERATION		4.210	High	0.27 - 4.2	μΙU/mL







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METHOD: ELECTROCHEMILUMINESCENCE

PAPANICOLAOU SMEAR

TEST METHOD CONVENTIONAL GYNEC CYTOLOGY

SPECIMEN TYPE P 1015/22

TWO UNSTAINED CERVICAL SMEARS RECEIVED

REPORTING SYSTEM 2014 BETHESDA SYSTEM FOR REPORTING CERVICAL CYTOLOGY

SPECIMEN ADEQUACY SATISFACTORY

THE SMEARS SHOW MAINLY SUPERFICIAL SQUAMOUS CELLS, FEW MICROSCOPY

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

MODERATE POLYMORPHS.

INTERPRETATION / RESULT NEGATIVE FOR INTRAEPITHELIAL LESION OR MALIGNANCY

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 LYPE B

METHOD: GEL COLUMN AGGLUTINATION METHOD.

POSITIVE RH LYPE

METHOD: GEL COLUMN AGGLUTINATION METHOD.

XRAY-CHEST

IMPRESSION NO ABNORMALITY DETECTED

TMT OR ECHO

TMT OR ECHO MILD POSITIVE

ECG

ECG WITHIN NORMAL LIMITS

MAMOGRAPHY (BOTH BREASTS)

MAMOGRAPHY 30TH BREASTS SIMPLE CYST IN RIGHT BREAST.

DENSE BREASTS.

MEDICAL HISTORY

HYPOTHYROID SINCE 13 YEARS. RELEVANT PRESENT HISTORY RELEVANT PAST HISTORY CHOLECYSTECTOMY IN 2011. H/O UTERINE FIBROIDS.

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

ALCOHOL:







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MENSTRUAL HISTORY (FOR FEMALES)	REG 24-32/2 DAYS.	
LMP (FOR FEMALES)	12/8/2022.	
OBSTETRIC HISTORY (FOR FEMALES)	2FTNDA2L2	

13 YEARS BACK.

HIGH BLOOD PRESSURE & DIABETES: BOTH PARENTS. RELEVANT FAMILY HISTORY

HISTORY OF MEDICATIONS NOT SIGNIFICANT

ANTHROPOMETRIC DATA & BMI

LCB (FOR FEMALES)

HEIGHT IN METERS 1.59 mts WEIGHT IN KGS. 69 Kgs 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 NORMAL

RESPIRATORY RATE

130/70 MM HG mm/Hg

(SUPINE) PERICARDIUM NORMAL APEX BEAT NORMAL HEART SOUNDS NORMAL



CARDIOVASCULAR SYSTEM

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MUDAMUDO	ADODNIT	
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 WITHIN NORMAL LIMIT DISTANT VISION LEFT EYE WITHOUT GLASSES WITHIN NORMAL LIMIT NEAR VISION RIGHT EYE WITHOUT GLASSES REDUCED VISUAL ACUITY N/8 NEAR VISION LEFT EYE WITHOUT GLASSES REDUCED VISUAL ACUITY N/12 NEAR VISION RIGHT EYE WITH GLASSES GLASSES NOT BROUGHT. NEAR VISION LEFT EYE WITH GLASSES GLASSES NOT BROUGHT.







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COLOUR VISION NORMAL

SUMMARY

RELEVANT HISTORY NOT SIGNIFICANT OVERWEIGHT: BMI 27 RELEVANT GP EXAMINATION FINDINGS

REDUCED ACUITY FOR NEAR VISION. REMARKS / RECOMMENDATIONS

1) CARDIOLOGIST CONSULT IN VIEW OF POSITIVE STREE TEST.

TO DO S, IRON STUDIES.

3) IRON RICH DIET ADVISED. ADD GREEN LEAFY VEGETABLES, DATES

BEETROOT TO THE DAILY DIET.

4) GYNAEC CONSULT FOR UTERINE FIBROIDS

 5) ANNUAL USG ABDOMEN/PELVIS & SONOMAMMOGRAPHY. 6) 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.

WBC DIFFERENTIAL COUNT - NLRThe 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 miedications.

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

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 proteinum 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 partium. 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 polkilocytosis, spherocytosis or sickle cells.

Nathan and Oski's Haematology of Infancy and Childhood, 5th edition
 Paediatric reference intervals. AACC Press, 7th edition. Edited by S. Soldin

3. The reference for the adult reference range is "Practical Haematology by Dacie and Lewis, 10th Edition" GLYCOSYLATED HEMOGLOBIN, EDTA WHOLE BLOOD-

Glycosylatec hemoglobin (GHb) has been firmly established as an index of long-term blood glucose concentrations 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,







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PATIENT NAME: SUPRIYA DIWAKAR

PATIENT ID: SUPRF030479L

ACCESSION NO: 0181VI000303 AGE: 43 Years SEX: Female

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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-spienectomy 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. Tretz Textbook of Clinical Chemistry and Molecular Diagnostics, edited by Carl A Burtis, Edward R. Ashwood, David E Bruns, 4th Edition, Elsevier publication, 2006,
- 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 75 grams of glucose in 300 ml water, over a period of 5 minutes.

LIVER FUNCTION PROFILE, SERUM-

LIVER FUNCTION PROFILE
Bilirubin is a yellowish pigment found in bile and is a breakdown product of normal heme catabolism. Bilirubin is excreted in bile and urine, and elevated levels may give yellow discoloration in jaundice. Elevated levels results from increased bilirubin production (eg, hemolysis and ineffective erythropolesis), 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, pancreaths, 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 hile 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 pandreas, 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, Malnutribon, 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 Increasec levels

Pre renal

- High protein diet, Increasec 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)



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Lower than normal level may be due to:

- Myasthenia Gravis
- Muscular dystrophy URIC ACID, SERUM-Causes of Increasec levels Dietary
- High Protein Intake.
- Prolonged Fasting,Rapid weight loss.

Gout

Lesch nyhan syndrome.

Type 2 DM.

Metabolic syndrome.

Causes of decreasec 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 alobulin

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), 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 influsion. 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

nypertuction, saincylate intoxication and with excessive infusion of isotonic saline or extremely high gletary intake of sait. Unlonde 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 prolonged vomiting,
THYROID PANEL, SERUMTriiodothyronine 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 (T5H), which is released from the pituitary gland. Elevated concentrations of T3, and T4 in the blood inhibit the production of T5H.

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 tree and biologically active.

In primary hypothyroidism, TSH levels are significantly elevated, while in secondary and tertiary 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 TOTAL T4 TSH3G TOTAL T3 Pregnancy (µg/dL) (μIU/mL) (ng/dL)

0.1 - 2.5 0.2 - 3.0 0.3 - 3.0 81 - 190 First Trimester 6.6 - 12.4100 - 260 100 - 260 2nd Trimester 6.6 - 15.5 6.6 - 15.53rc Trimester Below mentioned are the guidelines for age related reference ranges for T3 and T4.

(μg/dL) 1-3 day: 8.2 - 19.9 (ng/dL) New Born: 75 - 260 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.







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

Reference:

Relate a live:

1. Burbs 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

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.







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MEDI WHEEL FULL BODY HEALTH CHECKUP ABOVE 40FEMALE

ULTRASOUND ABDOMEN ULTRASOUND ABDOMEN GRADE | FATTY LIVER BULKY UTERUS WITH UTERINE FIBROIDE.

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

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