

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

SRLLtd

S.K. Tower,Hari Niwas, LBS Marg THANE, 400602 MAHARASHTRA, INDIA Tel: 9111591115, Fax: CIN - U74899PB1995PLC045956

Email: customercare.thane@srl.in

PATIENT NAME: PRAMOD SAGAR PATIENT ID: PRAMM050172181

ACCESSION NO: 0181VI000317 AGE: 50 Years SEX: Male

DRAWN: RECEIVED: 10/09/2022 09:53 REPORTED: 13/09/2022 15:19

REFERRING DOCTOR: SELF CLIENT PATIENT ID:

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

# MEDI WHEEL FULL BODY HEALTH CHECK UP ABOVE 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	13.2		13.0 - 17.0	g/dL
METHOD: SLS-HEMOGLOBIN DETECTION METHOD				
RED BLOOD CELL COUNT	4.99		4.5 - 5.5	mil/µL
METHOD: HYDRODYNAMIC FOCUSING BY DC DETECTION				
WHILE BLOOD CELL COUNT	6.01		4.0 - 10.0	thou/µL
METHOD: FLUORESCENCE FLOW CYTOMETRY				
PLATELET COUNT	278		150 - 410	thou/µL
METHOD: HYDRODYNAMIC FOCUSING BY DC DETECTION				
RBC AND PLATELET INDICES				
HEMATOCRIT	42.2		40.0 - 50.0	%
METHOD: CUMULATIVE PULSE HEIGHT DETECTION METHOD				
MEAN CORPUSCULAR VOL	84.6		83.0 - 101.0	tL
METHOD: CALCULATED FROM RBC & HCT				
MEAN CORPUSCULAR HGB.	26.5	Low	27.0 - 32.0	pg
METHOD: CALCULATED FROM THE RBC & HGB				
MEAN CORPUSCULAR HEMOGLOBIN	31.3	Low	31.5 - 34.5	g/dL
CONCENTRATION  METHOD: CALCULATED FROM THE HGB & HCT				
MENTZER INDEX	17.0			
RED CELL DISTRIBUTION WIDTH	17.6	High	11.6 - 14.0	%
METHOD : CALCULATED FROM RBC SIZE DISTRIBUTION CURVE		_		
MEAN PLATELET VOLUME	10.8		6.8 - 10.9	fL
METHOD: CALCULATED FROM PLATELET COUNT & PLATELET HEMAT	OCRIT			
CHEMICAL EXAMINATION, URINE				
P⊢	6.5		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 OXIDA	SE PEROXIDASE			
KETONES		NOT DETECTED	NOT DETECTED	
METHOD: NITROPRUSSIDE	REACTION			
BLOOD		NOT DETECTED	NOT DETECTED	
METHOD : PEROXIDASE		NORMAL	NORMA	
UROBILINOGEN	CLUDEACTION	NORMAL	NORMAL	
METHOD : MODIFIED EHRLI	CH REACTION	NOT DETECTED	NOT DETECTED	
NITRITE	DROBENZO(H)QUINOLIN-3-OL	NOT DETECTED	NOT DETECTED	
LEUKOCYTE ESTERASE		NOT DETECTED	NOT DETECTED	
WBC DIFFERENTIAL		NOI DETECTED	NOT DETECTED	
		F7	40 00	0/
SEGMENTED NEUTROP  METHOD: FLOW CYTOMETR'		57	40 - 80	%
ABSOLUTE NEUTROPH		3.45	2.0 - 7.0	thou/µL
METHOD : FLOW CYTOMETR		5.45	2.0 - 7.0	поаль
LYMPHOCYTES	T WITH BOTH SCAFFERING	35	20 - 40	%
METHOD: FLOW CYTOMETR	Y WITH LIGHT SCATTERING		20 10	,0
ABSOLUTE LYMPHOCYT		2.11	1.0 - 3.0	thou/µL
METHOD : FLOW CYTOMETR	Y WITH LIGHT SCATTERING			•
NEUTROPHIL LYMPHOC	YTE RATIC (NLR)	1.6		
EOSINOPHILS		3	1 - 6	%
METHOD : FLOW CYTOMETR	Y WITH LIGHT SCATTERING			
ABSOLUTE EOSINOPHI	IL COUNT	0.17	0.02 - 0.50	thou/µL
METHOD: FLOW CYTOMETR	Y WITH LIGHT SCATTERING			
MONOCYTES		5	2 - 10	%
METHOD : FLOW CYTOMETR				
ABSOLUTE MONOCYTE		0.27	0.2 - 1.0	thou/µL
METHOD : FLOW CYTOMETR		EDTA OMEAN		
DIFFERENTIAL COUNT		EDTA SMEAR		
MICROSCOPIC EXAM	IINATION, URINE			
PUS CELL (WBC'S)		1-2	0-5	/HPF
METHOD : MICROSCOPIC EX	KAMINATION	0.4	0.5	
EPITHELIAL CELLS	/AMAINIATION	0-1	0-5	/HPF
METHOD : MICROSCOPIC EX		NOT DETECTED	NOT DETECTED	/HPF
ERYTHROCYTES (RBC'S METHOD: MICROSCOPICE)	,	NOT DETECTED	NOT DETECTED	/ ITPT
CASTS	NAMINALION .	NOT DETECTED		
00013		NOT DETECTED		







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METHOD : MICROSCOPIC E	XAMINATION			
CRYSTALS		NOT DETECTED		
METHOD : MICROSCOPIC E	XAMINATION			
BACTERIA		NOT DETECTED	NOT DETECTED	
METHOD : MICROSCOPIC E	XAMINATION			
YEAST		NOT DETECTED	NOT DETECTED	
MORPHOLOGY				
RBC		ANISOCYTOSIS		
WBC		NORMAL MORPHOLOGY		
METHOD : MICROSCOPIC E	XAMINATION			
PLATELETS		ADEQUATE		
ERYTHRO SEDIMENT	TATION RATE, BLOOD			
SEDIMENTATION RAIL		06	0 - 14	mm at 1 hr
METHOD : WESTERGREN M				
GLYCOSYLATED HEN	OGLOBIN, EDTA WHOLE B	LOOD		
GLYCOSYLATED HEMC	OGLOBIN (HBA1C)	5.4	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 GLUCO METHOD : CALCULATED PAR		108.3	< 116.0	mg/dL
GLUCOSE, FASTING,	, PLASMA			
GLUCOSE, FASTING, F	PLASMA	85	Normal 75 - 99 Pre-diabetics: 100 - 125 Diabetic: > or = 126	mg/dL
METHOD : ENZYMATIC REF	ERENCE METHOD WITH HEXOKINASE			
GLUCOSE, POST-PRA	ANDIAL, PLASMA			
GLUCOSE, POST-PRAN	IDIAL, PLASMA	90	70 - 139	mg/dL
METHOD: ENZYMATIC REF	ERENCE METHOD WITH HEXOKINASE			
CORONARY RISK PR	OFILE, SERUM			
CHOLESTEROL		180	Desirable cholesterol level < 200 Borderline high cholesterol 200 - 239 High cholesterol > / = 240	mg/dL
METHOD : ENZYMATIC COL	ORIMETRIC ASSAY			

METHOD: ENZYMATIC COLORIMETRIC ASSAY



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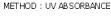
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TRIGLYCERIDES	115		Normal: < 150 Borderline high: 150 - 199 High: 200 - 499 Very High: >/= 500	mg/dL
METHOD : ENZYMATIC COLORIMETRIC ASSAY  HDL CHOLESTEROL	25.2	Low	Low HDL Cholesterol <40	mg/dL
TIDE CHOLESTEROL	23.2	LOW	EBW FIDE Cholesteror 140	mg/aL
METHOD - ENGRAPTIC COLODIMETRIC			High HDL Cholesterol >/= 60	
METHOD : ENZYMATIC, COLORIMETRIC	132	⊔iab	Adult loveler	ma/dl
CHOLESTEROL LDL	132	nigii	Adult levels: Optimal < 100 Near optimal/above optimal: 11 129 Borderline high: 130-159 High: 160-189 Very high: = 190	mg/dL 00-
METHOD: ENZYMATIC COLORIMETRIC ASSAY			. 0	
NON HDL CHOLESTEROL	155	High	Desirable: < 130 Above Desirable: 130 -159 Borderline High: 160 - 189 High: 190 - 219 Very high: > / = 220	mg/dL
CHOL/HDL RATIO	7.1	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	5.2	High	0.5 - 3.0 Desirable/Low Risk 3.1 - 6.0 Borderline/Moderate I >6.0 High Risk	Risk
VERY LOW DENSITY LIPOPROTEIN	23.0		< OR = 30.0	mg/dL
LIVER FUNCTION PROFILE, SERUM				
BILIRUBIN, TOTAL  METHOD: COLORIMETRIC DIAZO	0.34		Upto 1.2	mg/dL
BILIRUBIN, DIRECT	0.17		< 0.30	mg/dL
BILIRUBIN, INDIRECT	0.17		0.1 - 1.0	mg/dL
TOTAL PROTEIN  METHOD: COLORIMETRIC	7.1		6.0 - 8.0	g/dL
ALBUMIN METHOD: COLORIMETRIC	4.6		3.97 - 4.94	g/dL
GLOBULIN	2.5		2.0 - 3.5	g/dL
ALBUMIN/GLOBULIN RATIO	1.8		1.0 - 2.1	RATIO
ASPARTATE AMINOTRANSFERASE (AST/SGOT)  METHOD: UV AB SORBANCE	21		< OR = 50	U/L









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	CEEDAGE (ALT/CODT)	1.7		< OR = 50	1.171
ALANINE AMINOTRANS METHOD : UV AB SORBANCE	, , ,	17		< OR = 30	U/L
ALKALINE PHOSPHATA		90		40 - 129	U/L
METHOD : COLORIMETRIC					-, -
GAMMA GLUTAMYL TR	ANSFERASE (GGT)	17		0 - 60	U/L
METHOD : ENZYMATIC, COL	ORIMETRIC				
LACTATE DEHYDROGE		182		125 - 220	U/L
METHOD: UV ABSORBANCE					
SERUM BLOOD UREA		_			
BLOOD UREA NITROGE		6		6 - 20	mg/dL
METHOD : ENZYMATIC ASSA					
CREATININE, SERUN CREATININE	VI	0.91		0.7 - 1.2	mg/dL
METHOD : COLORIMETRIC		0.91		0.7 - 1.2	mg/ac
BUN/CREAT RATIO					
BUN/CREAT RATIO		6.59	Low	8.0 - 15.0	
URIC ACID, SERUM					
URIC ACID		5.3		3.4 - 7.0	mg/dL
METHOD : ENZYMATIC COL	ORIMETRIC ASSAY				
TOTAL PROTEIN, SE	RUM				
TOTAL PROTEIN		7.1		6.0 - 8.0	g/dL
METHOD: COLORIMETRIC					
ALBUMIN, SERUM					
ALBUMIN		4.6		3.97 - 4.94	g/dL
METHOD : COLORIMETRIC					
GLOBULIN					7.0
GLOBULIN	######################################	2.5		2.0 - 3.5	g/dL
ELECTROLYTES (NA,	/K/CL), SERUM		_		
SODIUM		132		136 - 145	mmol/L
POTASSIUM		5.38	High	3.5 - 5.1	mmol/L
CHLORIDE		99		98 - 107	mmol/L
THYROID PANEL, SE	RUM				
T3		62.1	Low	80 - 200	ng/dL
METHOD : ELECTROCHEMIL	UMINESCENCE	FDF		E 1	الدليدان
T4  METHOD: ELECTROCHEMIL	LIMINECCENICE	5.35		5.1 - 14.1	µg/dL
TSH 3RD GENERATION		1.480		0.27 - 4.2	μΙU/mL
TOTTOND GENERALION		1.400		0.27 - 4.2	μιο/πι







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

METHOD: ELECTROCHEMILUMINESCENCE

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

**IMPRESSION** NO ABNORMALITY DETECTED

TMT OR ECHO

TMT OR ECHO 2D ECHO :-

Structurally normal valves, Mild TR

No RWMA.

Good Left Ventricular systolic function. LVEF 60%

Normal LV Diastolic function. No e/o pulmonary hypertension

**ECG** 

ECG WITHIN NORMAL LIMITS

MEDICAL HISTORY

RELEVANT PRESENT HISTORY NOT SIGNIFICANT

RELEVANT PAST HISTORY H/O PILES HAS CONSULTED A SURGEON ON TREATMENT.

COVID 2 YEARS BACK, HOME QUARANRINED.

RELEVANT PERSONAL HISTORY MARRIED / 3 CHILD / MIXED DIET / NO ALLERGIES / NO SMOKING /

OCC ALCOHOL NOT SIGNIFICANT

RELEVANT FAMILY HISTORY HISTORY OF MEDICATIONS NOT SIGNIFICANT

ANTHROPOMETRIC DATA & BMI

HEIGHT IN METERS 1.68 mts WEIGHT IN KGS. 85 Kgs

BMI BMI & Weight Status as follows: kg/sqmts 30

Below 18.5: Underweight 18.5 - 24.9: Normal 25.0 - 29.9: Overweight 30.0 and Above: Obeše

GENERAL EXAMINATION

MENTAL / EMOTIONAL STATE NORMAL PHYSICAL ATTITUDE NORMAL GENERAL APPEARANCE / NUTRITIONAL STATUS OBESE BUILT / SKELETAL FRAMEWORK **AVERAGE** FACIAL APPEARANCE NORMAL



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OLTA	NORMA	
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	68/MIN.REGULAR, A BRUIT	ALL PERIPHERAL PULSES WELL FELT, NO CAROTID
RESPIRATORY RATE	NORMAL	
CARDIOVASCULAR SYSTEM		
BP	130/80 MM HG	mm/Hg
	(SUPINE)	,
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 (NORMA	AL)
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	
CENCODY CYCTEM	NODMAL	

NORMAL



SENSORY SYSTEM

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VIE. 2. 11 2. 13 2 3 3 1 3 1 1 1 1 2 1 3 1 3 1 3 1 3	3			
Test Report Status <b><u>Preliminary</u></b>	Results	Biological Reference Interval	Units	
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 ACUIT	ΓY 6/12		
DISTANT VISION LEFT EYE WITHOUT GLASSES	REDUCED VISUAL ACUIT	ΓY 6/9		
DISTANT VISION RIGHT EYE WITH GLASSES	GLASSES NOT BROUGH	Т.		
DISTANT VISION LEFT EYE WITH GLASSES	GLASSES NOT BROUGH	Т.		
NEAR VISION RIGHT EYE WITHOUT GLASSES	REDUCED VISUAL ACUIT	ΓΥ n/0		
NEAR VISION LEFT EYE WITHOUT GLASSES	REDUCED VISUAL ACUIT	ΓΥ n/0		
NEAR VISION RIGHT EYE WITH GLASSES	GLASSES NOT BROUGH	Т.		
NEAR VISION LEFT EYE WITH GLASSES	GLASSES NOT BROUGH	Т.		
COLOUR VISION	COLOUR BLIND: 03/17			
SUMMARY	RESULT PENDING			

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-

Microscopie: Examination, ording-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.







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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, 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 redicells 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 rec blood cell (average 120 days) and the blood glucose concentration. Because the rate of formation of GHb is directly proportional to the concentration of glucose in the blood,

the GHb concentration represents the integrated values for glucose over the preceding 6-8 weeks.

Any condition that alters the life span of the red blood cells has the potential to alter the GHb level. Samples from patients with hemolytic anemias will exhibit decreased glycated hemoglobin values due to the shortened life span of the red cells. This effect will depend upon the severity of the anemia. Samples from patients with polycythemia

or post-splenectomy may exhibit increased glycated hemoglobin values due to a somewhat longer life span of the red cells.
Glycosylated hemoglobins results from patients with HbSS, HbCC, and HbSC and HbD must be interpreted with caution, given the pathological processes, including anemia, increased recipied recipied in the conditions, alternative forms of testing such 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."

- 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,
- 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-PRANDÏAL, 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 tounc 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. Elevated levels results from increased bilirubin production (eg, hemolysis and ineffective erythropoiesis), decreased bilirubin excretion (eg, obstruction and hepatitis), and abnormal bilirubin metabolism (eg, hereditary and neonatal jaundice). Conjugated (direct) bilirubin is elevated more than unconjugated (indirect) bilirubin in Viral hepatitis, Drug reactions, Alcoholic liver disease Conjugated (direct) bilirubin is also elevated more than unconjugated (indirect) bilirubin when there is some kind of blockage of the bile ducts like in Gallstones getting into the bile ducts, tumors & Scarring of the bile ducts. Increased unconjugated (indirect) bilirubin may be a result of Hemolytic or pernicious anemia, Transfusion reaction & a common metabolic condition termed Gilbert syndrome, due to low levels of the enzyme that attaches sugar molecules to bilirubin.
AST is an enzyme found in various parts of the body. AST is found in the liver, heart, skeletal muscle, kidneys, brain, and red blood cells, and it is commonly measured

clinically as a marker for liver health. AST levels increase during chronic viral hepatitis, blockage of the bile duct, cirrhosis of the liver, liver cancer, kidney failure, hemolytic anemia, pancreatitis, hemochromatosis. AST levels may also increase after a heart attack or strenuous activity. ALT test measures the amount of this enzyme in the blood. ALT is found mainly in the liver, but also in smaller amounts in the kidneys, heart, muscles, and pancreas. It is commonly measured as a part of a diagnostic evaluation of hepatocellular injury, to determine liver health AST levels increase during acute hepatitis, sometimes due to a viral infection, ischemia to the liver, chronic hepatitis, obstruction of bile ducts, cirrhosis.

ALP is a protein found in almost all body tissues. Tissues with higher amounts of ALP include the liver, bile ducts and bone. Elevated ALP levels are seen in Biliary obstruction, Osteoblastic bone tumors, osteomalacia, hepatitis, Hyperparathyroidism, Leukemia, Lymphoma, Paget's disease,Rickets,Sarcoidosis etc. Lower-than-normal ALP levels seen in Hypophosphatasia,Malnutrition,Protein deficiency,Wilson's disease.GGT is an enzyme found in cell membranes of many tissues mainly in the liver,kidney and pancreas.It is also found in other tissues including intestine, spleen, heart, brain and seminal vesicles. The highest concentration is in the kidney, but the liver is considered the source of normal enzyme activity. Serum GGT has been widely used as an index of liver dysfunction. Elevated serum GGT activity can be found in diseases of the liver, billiary system normal enzyme activity, section of in his been widely used as all moex or liver dystuction, let enzyme activity, section of in his been widely used as all moex or liver dystuction, let enzyme activity, section of interests entering the constructive 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, indicating HIV and hepatitis B or C, Multiple myelom a, Waldenstrom's disease, Lower-than-normal levels may be due to: Agammaglobulinemia, Bleeding (hemorrhage), Burns, Glomerulonephritis, Liver Biocases, 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







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PATIENT ID:

PRAMM050172181

Email: customercare.thane@srl.in

PATIENT NAME: PRAMOD SAGAR

ACCESSION NO: 0181VI000317 AGE: 50 Years SEX: Male

DRAWN: RECEIVED: 10/09/2022 09:53 REPORTED: 13/09/2022 15:19

REFERRING DOCTOR: SELF CLIENT PATIENT ID:

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

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 databolism, 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
- 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
- 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 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 (hyperchloremia metabolic acidosis), acute renal failure, metabolic acidosis associated with prolonged diarrhea and loss of sodium bicarbonated, diabetes inspidus, adrenocorbical hypertuction, salicylate intoxication and with excessive intusion of isotionic 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



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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 caried in production. Most of the dryloid institution of t 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

(μIU/mL) 0.1 - 2.5 0.2 - 3.0 0.3 - 3.0 (ng/dL) 81 - 190 100 - 260 Pregnancy First Trimester (µg/dL) 6.6 - 12.4 2nd Trimester 6.6 - 15.5 6.6 - 15.5100 - 2603rc Trimester

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

(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 or the report under biological reference range.

- 1. Burts 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. 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.

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EXAMINATIONS AND INVESTIGATIONS HAVE BEEN CONDUCTED BY OUR PANEL OF DOCTORS.



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# MEDI WHEEL FULL BODY HEALTH CHECK UP ABOVIR BEOLIMAPIEN DING

ULTRASOUND ABDOMEN

RESULT PENDING

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

Bhinchkhede

Dr.Priyal Chinchkhede Consultant Pathologist

Dr. Ushma Wartikar Consultant Pathologist Dr.(Mrs)Neelu K Bhojani

Lab Head





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