Patient Ref. No. 31000004501404

Cert. No. MC-2396

CLIENT CODE : C000138363

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

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Salt Lake,
KOLKATA, 700091
WEST BENGAL, INDIA
Tel : 9111591115,
CIN - U74899PB1995PLC045956
Email : customercare.saltlake@srl.in

PATIENT NAME : DEBANGSHU SAMAJDAR PATIENT ID : **DEBAM13107931**

ACCESSION NO : **0031VI015678** AGE : 42 Years SEX : Male ABHA NO :

DRAWN : 19-09-2022 10:09 RECEIVED : 19-09-2022 10:18 REPORTED : 20-09-2022 15:06

REFERRING DOCTOR : SELF CLIENT PATIENT ID :

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

MEDI WHEEL FULL BODY HEALTH CHECK UP ABOVE 40 MALE

BLOOD COUNTS,EDTA WHOLE BLOOD

HEMOGLOBIN	**17.5**	**High**	13.0 - 17.0	g/dL
METHOD : SPECTROPHOTOMETRY				
RED BLOOD CELL COUNT	**5.93**	**High**	4.5 - 5.5	mil/µL
METHOD : ELECTRICAL IMPEDANCE				
WHITE BLOOD CELL COUNT	7.98		4.0 - 10.0	thou/µL
METHOD : ELECTRICAL IMPEDANCE				
PLATELET COUNT	170		150 - 410	thou/µL
METHOD : ELECTRONIC IMPEDENCE & MICROSCOPY				

RBC AND PLATELET INDICES

HEMATOCRIT	**53.4**	**High**	40 - 50	%
METHOD : CALCULATED				
MEAN CORPUSCULAR VOL	89.9		83 - 101	fL
METHOD : ELECTRICAL IMPEDANCE				
MEAN CORPUSCULAR HGB.	29.5		27.0 - 32.0	pg
METHOD : CALCULATED				
MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION	32.8		31.5 - 34.5	g/dL
METHOD : CALCULATED				
MENTZER INDEX	15.2			
RED CELL DISTRIBUTION WIDTH	**14.3**	**High**	11.6 - 14.0	%
METHOD : ELECTRICAL IMPEDANCE				
MEAN PLATELET VOLUME	9.3		6.8 - 10.9	fL
METHOD : CALCULATED				

WBC DIFFERENTIAL COUNT - NLR

SEGMENTED NEUTROPHILS	71		40 - 80	%
METHOD : FLOWCYTOMETRY, ELECTRONIC IMPEDANCE & MICROSCOPY.				
ABSOLUTE NEUTROPHIL COUNT	5.67		2.0 - 7.0	thou/µL
METHOD : FLOWCYTOMETRY & CALCULATED				
LYMPHOCYTES	20		20 - 40	%
METHOD : FLOWCYTOMETRY, ELECTRONIC IMPEDANCE & MICROSCOPY.				
ABSOLUTE LYMPHOCYTE COUNT	1.60		1 - 3	thou/µL
METHOD : FLOWCYTOMETRY & CALCULATED				
NEUTROPHIL LYMPHOCYTE RATIO (NLR)	3.5			
EOSINOPHILS	1		1 - 6	%

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ABSOLUTE EOSINOPHIL COUNT	0.08		0.02 - 0.50	thou/µL
METHOD : FLOWCYTOMETRY & CALCULATED				
MONOCYTES	8		2 - 10	%
METHOD : FLOWCYTOMETRY, ELECTRONIC IMPEDANCE & MICROSCOPY.				
ABSOLUTE MONOCYTE COUNT	0.64		0.20 - 1.00	thou/µL
METHOD : FLOWCYTOMETRY & CALCULATED				
BASOPHILS	0		0 - 2	%
METHOD : FLOWCYTOMETRY, ELECTRONIC IMPEDANCE & MICROSCOPY.				
ABSOLUTE BASOPHIL COUNT	**0**	**Low**	0.02 - 0.10	thou/µL
METHOD : FLOWCYTOMETRY & CALCULATED				
MORPHOLOGY				
RBC	NORMOCYTIC NORMOCHROMIC			
METHOD : MICROSCOPIC EXAMINATION				
WBC	NORMAL MORPHOLOGY			
METHOD : MICROSCOPIC EXAMINATION				
PLATELETS	ADEQUATE			
METHOD : MICROSCOPIC EXAMINATION				

Comments

NOTE- HB VALUE HAS BEEN RECHEKED , PLEASE CORRELATE CLINICALLY

ERYTHRO SEDIMENTATION RATE, BLOOD				
SEDIMENTATION RATE (ESR)	**17**	**High**	0 - 14	mm at 1 hr
METHOD : AUTOMATED (PHOTOMETRICAL CAPILLARY STOPPED FLOW KINETIC ANALYSIS)"				
GLYCOSYLATED HEMOGLOBIN, EDTA WHOLE BLOOD				
GLYCOSYLATED HEMOGLOBIN (HBA1C)	**12.3**	**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	**306.3**	**High**	< 116.0	mg/dL

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SRL LIMITED - KOLKATA REF. LAB **PATIENT REP**
Bio-Rad Variant II Turbo CDM 5.4 S/N : 13466 **V2TURBO_A1c**

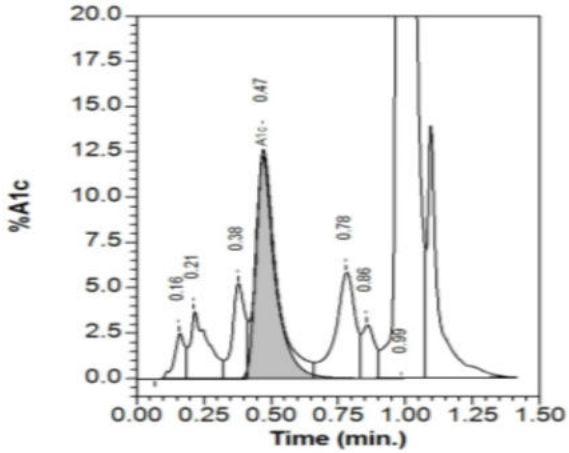
Patient Data		Analysis Data	
Sample ID:	3106477550	Analysis Performed:	19/09/2022 13:52:14
Patient ID:	0031VI015678	Injection Number:	4309
Name:	DEBANGSHUSAMAJDAR	Run Number:	316
Physician:		Rack ID:	
Sex:		Tube Number:	6
DOB:		Report Generated:	19/09/2022 14:07:14
		Operator ID:	

Comments:

Peak Name	NGSP %	Area %	Retention Time (min)	Peak Area
A1a	---	1.2	0.157	33556
A1b	---	3.0	0.212	86125
LA1c	---	3.2	0.377	93699
A1c	12.3*	---	0.469	307822
P3	---	5.2	0.778	148650
P4	---	1.7	0.855	48601
Ao	---	75.1	0.986	2165101

*Values outside of expected ranges Total Area: 2,883,554

HbA1c (NGSP) = 12.3* %



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Comments

FOR HbA1C
NOTE: INCREASED LEVELS OF GLYCOSYLATED HEMOGLOBIN MAY NEED CLINICAL CORRELATION . HIGH GLYCOSYLATED HEMOGLOBIN LEVELS MAY BE OBSERVED IN CONDITIONS SUCH AS UNCONTROLLED DIABETES, POOR COMPLIANCE WITH ANTIDIABETIC THERAPY, CHRONIC RENAL FAILURE, HYPERTRIGLYCERIDEMIA, IRON DEFICIENCY ANAEMIA, SALICYLATE THERAPY, HAEMOGLOBINOPATHIES LIKE THALASSAEMIA MAY ALSO SHOW HIGH GLYCOSYLATED HEMOGLOBIN LEVELS.

GLUCOSE, FASTING, PLASMA				
GLUCOSE, FASTING, PLASMA	**249**	**High**	74 - 100	mg/dL
METHOD : ENZYMATIC (HEXOKINASE/G-6-PDH)				
GLUCOSE, POST-PRANDIAL, PLASMA				
GLUCOSE, POST-PRANDIAL, PLASMA	**315**	**High**	140 Normal 140 - 199 Pre-diabetic > or = 200 Diabetic	mg/dL
METHOD : ENZYMATIC (HEXOKINASE/G-6-PDH)				
CORONARY RISK PROFILE, SERUM				
CHOLESTEROL	117		< 200 Desirable 200 - 239 Borderline High >/= 240 High	mg/dL
METHOD : ENZYMATIC ASSAY				
TRIGLYCERIDES	86		< 150 Normal 150 - 199 Borderline High 200 - 499 High >/=500 Very High	mg/dL
METHOD : GLYCEROL PHOSPHATE OXIDASE				
HDL CHOLESTEROL	**36**	**Low**	Low : < 40 High : > / = 60	mg/dL
METHOD : ACCELERATOR SELECTIVE DETERGENT METHODOLOGY				
CHOLESTEROL LDL	64			mg/dL
NON HDL CHOLESTEROL	81		Desirable: Less than 130 Above Desirable: 130-159 Borderline High: 160-189 High: 190 -219 Very High: >or = 220	mg/dL
METHOD : CALCULATED				
CHOL/HDL RATIO	3.3			
LDL/HDL RATIO	1.8			
VERY LOW DENSITY LIPOPROTEIN	17.2			mg/dL
LIVER FUNCTION PROFILE, SERUM				
BILIRUBIN, TOTAL	1.06		0.2 - 1.2	mg/dL

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METHOD : DIAZONIUM SALT			
BILIRUBIN, DIRECT	0.46	0.0 - 0.5	mg/dL
METHOD : DIAZO REACTION			
BILIRUBIN, INDIRECT	0.60	0.1 - 1.0	mg/dL
METHOD : CALCULATED			
TOTAL PROTEIN	7.6	6.0 - 8.30	g/dL
METHOD : BIURET			
ALBUMIN	4.2	3.5 - 5.2	g/dL
METHOD : COLORIMETRIC (BROMCRESOL GREEN)			
GLOBULIN	3.4	2.0 - 3.5	g/dL
ALBUMIN/GLOBULIN RATIO	1.2	1 - 2.1	RATIO
METHOD : CALCULATED PARAMETER			
ASPARTATE AMINOTRANSFERASE (AST/SGOT)	25	5 - 34	U/L
METHOD : ENZYMATIC (NADH (WITHOUT P-5'-P)			
ALANINE AMINOTRANSFERASE (ALT/SGPT)	35	0 - 55	U/L
METHOD : ENZYMATIC (NADH (WITHOUT P-5'-P)			
ALKALINE PHOSPHATASE	112	40 - 150	U/L
METHOD : PARA-NITROPHENYL PHOSPHATE			
GAMMA GLUTAMYL TRANSFERASE (GGT)	17	11 - 59	U/L
METHOD : L-GAMMA-GLUTAMYL-4-NITROANALIDE /GLYCYLGLYCINE KINETIC METHOD			
LACTATE DEHYDROGENASE	158	125 - 220	U/L
METHOD : IFCC LACTATE TO PYRUVATE			
SERUM BLOOD UREA NITROGEN			
BLOOD UREA NITROGEN	15	8.9 - 20.6	mg/dL
METHOD : UREASE METHOD			
CREATININE, SERUM			
CREATININE	1.16	0.72 - 1.25	mg/dL
METHOD : KINETIC ALKALINE PICRATE			
BUN/CREAT RATIO			
BUN/CREAT RATIO	12.93	5.0 - 15.0	
URIC ACID, SERUM			
URIC ACID	5.3	3.5 - 7.2	mg/dL
METHOD : URICASE			
TOTAL PROTEIN, SERUM			
TOTAL PROTEIN	7.6	6.0 - 8.3	g/dL
METHOD : BIURET			

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ALBUMIN, SERUM				
ALBUMIN	4.2		3.5 - 5.2	g/dL
METHOD : COLORIMETRIC (BROMCRESOL GREEN)				
GLOBULIN				
GLOBULIN	3.4		2.0 - 3.5	g/dL
METHOD : CALCULATED PARAMETER				
ELECTROLYTES (NA/K/CL), SERUM				
SODIUM	**135**	**Low**	136 - 145	mmol/L
METHOD : ION SELECTIVE ELECTRODE TECHNOLOGY INDIRECT				
POTASSIUM	3.80		3.5 - 5.1	mmol/L
METHOD : ION SELECTIVE ELECTRODE TECHNOLOGY INDIRECT				
CHLORIDE	99		98 - 107	mmol/L
METHOD : ION SELECTIVE ELECTRODE TECHNOLOGY INDIRECT				
PHYSICAL EXAMINATION, URINE				
COLOR	PALE YELLOW			
APPEARANCE	CLEAR			
SPECIFIC GRAVITY	1.015		1.003 - 1.035	
METHOD : DIPSTICK				
CHEMICAL EXAMINATION, URINE				
PH	6.0		4.7 - 7.5	
PROTEIN	NOT DETECTED		NOT DETECTED	
METHOD : DIPSTICK				
GLUCOSE	**DETECTED (++)**		NOT DETECTED	
METHOD : DIPSTICK				
KETONES	NOT DETECTED		NOT DETECTED	
METHOD : DIPSTICK				
BLOOD	NOT DETECTED		NOT DETECTED	
METHOD : DIPSTICK				
BILIRUBIN	NOT DETECTED		NOT DETECTED	
METHOD : DIPSTICK				
UROBILINOGEN	NORMAL		NORMAL	
METHOD : DIPSTICK				
NITRITE	NOT DETECTED		NOT DETECTED	
METHOD : DIPSTICK				
LEUKOCYTE ESTERASE	**DETECTED (+)**		NOT DETECTED	
MICROSCOPIC EXAMINATION, URINE				

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PUS CELL (WBC'S)	**5-7**	0-5	/HPF
EPITHELIAL CELLS	2-3	0-5	/HPF
ERYTHROCYTES (RBC'S)	NOT DETECTED	NOT DETECTED	/HPF
CASTS	NOT DETECTED		
CRYSTALS	NOT DETECTED		
BACTERIA	NOT DETECTED	NOT DETECTED	
YEAST	NOT DETECTED	NOT DETECTED	

Comments

URINALYSIS: MICROSCOPIC EXAMINATION IS CARRIED OUT ON CENTRIFUGED URINARY SEDIMENT.

NOTE: URINE GLUCOSE RECHECKED AND CONFIRMED BY BENEDICT''''S TEST.

THYROID PANEL, SERUM

T3	100.0	35 - 193	ng/dL
METHOD : TWO-STEP CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY			
T4	9.59	4.87 - 11.71	µg/dL
METHOD : TWO-STEP CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY			
TSH 3RD GENERATION	2.638	0.350 - 4.940	µIU/mL
METHOD : TWO-STEP CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY			

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

ABO GROUP	TYPE A
METHOD : GEL CARD METHOD	
RH TYPE	POSITIVE
METHOD : GEL CARD METHOD	

XRAY-CHEST

IMPRESSION NO ABNORMALITY DETECTED

TMT OR ECHO

TMT OR ECHO Echo done- Concentric LVH

ECG

ECG Possible lateral wall ischaemia in ecg

MEDICAL HISTORY

RELEVANT PRESENT HISTORY	Hypertension, Diabetes, Raised cholesterol on medication
RELEVANT PAST HISTORY	NOT SIGNIFICANT
RELEVANT PERSONAL HISTORY	Smoker-8/day
RELEVANT FAMILY HISTORY	Father - Diabetes, Mother - Hypertension

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OCCUPATIONAL HISTORY	NOT SIGNIFICANT		
HISTORY OF MEDICATIONS	NOT SIGNIFICANT		
ANTHROPOMETRIC DATA & BMI			
HEIGHT IN METERS	1.66		mts
WEIGHT IN KGS.	97		Kgs
BMI	35	BMI & Weight Status as follows: Below 18.5: Underweight 18.5 - 24.9: Normal 25.0 - 29.9: Overweight 30.0 and Above: Obese	kg/sqmts
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	78/min- REGULAR, ALL PERIPHERAL PULSES WELL FELT		
RESPIRATORY RATE	NORMAL		
CARDIOVASCULAR SYSTEM			
BP	120/80 mm Hg		mm/Hg
PERICARDIUM	NORMAL		
APEX BEAT	NORMAL		
HEART SOUNDS	NORMAL		
MURMURS	ABSENT		
RESPIRATORY SYSTEM			
SIZE AND SHAPE OF CHEST	NORMAL		
MOVEMENTS OF CHEST	SYMMETRICAL		

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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 WITH GLASSES	6/6		
DISTANT VISION LEFT EYE WITH GLASSES	6/15		
NEAR VISION RIGHT EYE WITH GLASSES	N10		
NEAR VISION LEFT EYE WITH GLASSES	N10		
COLOUR VISION	NORMAL		
BASIC ENT EXAMINATION			
EXTERNAL EAR CANAL	NORMAL		
TYMPANIC MEMBRANE	NORMAL		
NOSE	NO ABNORMALITY DETECTED		

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PATIENT NAME : DEBANGSHU SAMAJDAR PATIENT ID : **DEBAM13107931**

ACCESSION NO : **0031VI015678** AGE : 42 Years SEX : Male ABHA NO :

DRAWN : 19-09-2022 10:09 RECEIVED : 19-09-2022 10:18 REPORTED : 20-09-2022 15:06

REFERRING DOCTOR : SELF CLIENT PATIENT ID :

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

SINUSES	CLEAR
THROAT	NO ABNORMALITY DETECTED
TONSILS	NOT ENLARGED
BASIC DENTAL EXAMINATION	
TEETH	NORMAL
GUMS	HEALTHY
SUMMARY	
RELEVANT HISTORY	Hypertension, Diabetes, Raised cholesterol on medication
RELEVANT GP EXAMINATION FINDINGS	Obese (97 kg)
RELEVANT LAB INVESTIGATIONS	Raised HbA1c (12.3), FBS (249), PPBS (315), low sodium (135), glucose ++ in urine, Pus cells and Leukocyte esterase in urine
RELEVANT NON PATHOLOGY DIAGNOSTICS	Hepatomegaly with grade I fatty liver in usg Concentric LVH in echo Possible lateral wall ischaemia in ecg
REMARKS / RECOMMENDATIONS	On examination and investigations the candidate is found to be obese, hypertensive, diabetic and has raised HbA1c (12.3), FBS (249), PPBS (315), low sodium (135), glucose ++ in urine, Pus cells and Leukocyte esterase in urine Hepatomegaly with grade I fatty liver in usg Concentric LVH in echo Possible lateral wall ischaemia in ecg Should follow the given advice: 1. Salt restricted diabetic diet 2. Reduce body weight 3. Estimated body weight should be : 66 kg 4. Regular physical exercise and walking 5. Avoid fat and oily diet 6. Urine for C/S 7. Follow up with physician and Opthalmologist.

Comments

MEDICAL EXAMINATION REVIEWED BY:

DR. DEBIKA ROY, MBBS
REG NO: 51651 (WBMC)
CONSULTANT PHYSICIAN
WELLNESS CLINIC
SALT LAKE REF LAB, KOLKATA

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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.
RBC AND PLATELET INDICES-
Mentzer index (MCV/RBC) is an automated cell-counter based calculated screen tool to differentiate cases of Iron deficiency anaemia(>13) from Beta thalassaemia trait (<13) in patients with microcytic anaemia. This needs to be interpreted in line with clinical correlation and suspicion. Estimation of HbA2 remains the gold standard for diagnosing a case of beta thalassaemia trait.
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.
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"
GLYCOSYLATED HEMOGLOBIN, EDTA WHOLE BLOOD-
Glycosylated hemoglobin (GHb) has been firmly established as an index of long-term blood glucose concentrations and as a measure of the risk for the development of complications in patients with diabetes mellitus. Formation of GHb is essentially irreversible, and the concentration in the blood depends on both the life span of the red blood cell (average 120 days) and the blood glucose concentration. Because the rate of formation of GHb is directly proportional to the concentration of glucose in the blood, the GHb concentration represents the integrated values for glucose over the preceding 6-8 weeks.
Any condition that alters the life span of the red blood cells has the potential to alter the GHb level. Samples from patients with hemolytic anemias will exhibit decreased glycated hemoglobin values due to the shortened life span of the red cells. This effect will depend upon the severity of the anemia. Samples from patients with polycythemia or post-splenectomy may exhibit increased glycated hemoglobin values due to a somewhat longer life span of the red cells.
Glycosylated hemoglobins results from patients with 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 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 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 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

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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,biliary system and pancreas.Conditions that increase serum GGT are obstructive liver disease,high alcohol consumption and use of enzyme-inducing drugs etc.Serum total protein,also known as total protein,is a biochemical test for measuring the total amount of protein in serum.Protein in the plasma is made up of albumin and globulin.Higher-than-normal levels may be due to:Chronic inflammation or infection,including HIV and hepatitis B or C,Multiple myeloma,Waldenstrom's disease.Lower-than-normal levels may be due to: Agammaglobulinemia,Bleeding (hemorrhage),Burns,Glomerulonephritis,Liver disease, Malabsorption,Malnutrition,Nephrotic syndrome,Protein-losing enteropathy etc.Human serum albumin is the most abundant protein in human blood plasma.It is produced in the liver.Albumin constitutes about half of the blood serum protein.Low blood albumin levels (hypoalbuminemia) can be caused by:Liver disease like cirrhosis of the liver, nephrotic syndrome,protein-losing enteropathy,Burns,hemodilution,increased vascular permeability or decreased lymphatic clearance,malnutrition and wasting etc
SERUM BLOOD UREA NITROGEN-
Causes of Increased levels
Pre renal
• High protein diet, Increased protein catabolism, GI haemorrhage, Cortisol, Dehydration, CHF Renal
• Renal Failure
Post Renal
• Malignancy, Nephrolithiasis, Prostatism

Causes of decreased levels
• Liver disease
• SIADH.
CREATININE, SERUM-
Higher than normal level may be due to:
• Blockage in the urinary tract
• Kidney problems, such as kidney damage or failure, infection, or reduced blood flow
• Loss of body fluid (dehydration)
• Muscle problems, such as breakdown of muscle fibers
• Problems during pregnancy, such as seizures (eclampsia)), or high blood pressure caused by pregnancy (preeclampsia)

Lower than normal level may be due to:
• Myasthenia Gravis
• Muscular dystrophy
URIC ACID, SERUM-
Causes of Increased levels
Dietary
• High Protein Intake.
• Prolonged Fasting,
• Rapid weight loss.
Gout
Lesch nyhan syndrome.
Type 2 DM.
Metabolic syndrome.

Causes of decreased levels
• Low Zinc Intake
• OCP's
• Multiple Sclerosis

Nutritional tips to manage increased Uric acid levels
• Drink plenty of fluids
• Limit animal proteins
• High Fibre foods
• Vit C Intake
• Antioxidant rich foods
TOTAL PROTEIN, SERUM-
Serum total protein,also known as total protein, is a biochemical test for measuring the total amount of protein in serum..Protein in the plasma is made up of albumin and globulin

Higher-than-normal levels may be due to: Chronic inflammation or infection, including HIV and hepatitis B or C, Multiple myeloma, Waldenstrom's disease
Lower-than-normal levels may be due to: Agammaglobulinemia, Bleeding (hemorrhage),Burns,Glomerulonephritis, Liver disease, Malabsorption, Malnutrition, Nephrotic syndrome,Protein-losing enteropathy etc.

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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 (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, Addisonian crisis, certain types of metabolic acidosis, persistent gastric secretion and prolonged vomiting,
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
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

Levels in Pregnancy	TOTAL T4 (µg/dL)	TSH3G (µIU/mL)	TOTAL T3 (ng/dL)
First Trimester	6.6 - 12.4	0.1 - 2.5	81 - 190
2nd Trimester	6.6 - 15.5	0.2 - 3.0	100 - 260
3rd Trimester	6.6 - 15.5	0.3 - 3.0	100 - 260

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

	T3 (ng/dL)	T4 (µg/dL)
New Born:	75 - 260	1-3 day: 8.2 - 19.9
.		1 Week: 6.0 - 15.9

NOTE: TSH concentrations in apparently normal euthyroid subjects are known to be highly skewed, with a strong tailed distribution towards higher TSH values. This is well documented in the pediatric population including the infant age group.
Kindly note: Method specific reference ranges are appearing on the report under biological reference range.

Reference:
1. Burtis 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: "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."

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The test is performed by both forward as well as reverse grouping methods.
MEDICAL
HISTORY-**********
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MEDI WHEEL FULL BODY HEALTH CHECK UP ABOVE 40 MALE

ULTRASOUND ABDOMEN

ULTRASOUND ABDOMEN

Hepatomegaly with grade I fatty liver.

****End Of Report****
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Dr. Chaitali Ray, PhD	Dr.Anwesha Chatterjee,MD	Dr.Himadri Mondal, MD	Dr. Debika Roy
Chief Biochemist cum MRQA	Pathologist	Consultant Microbiologist	MBBS Consultant Physician

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