



CLIENT CODE: C000138379
CLIENT'S NAME AND ADDRESS:

ACROFEMI HEALTHCARE LTD (MEDIWHEEL) F-703, LADO SARAI, MEHRAULI SOUTH WEST DELHI

SOUTH WEST DELHI NEW DELHI 110030 DELHI INDIA 8800465156 SRL Ltd PLOT No. 88, ROAD No. 15,MIDC ESTATE,ANDHERI (EAST) MUMBAI, 400093

MAHARASHTRA, INDIA

Tel: 09152729959/9111591115, CIN - U74899PB1995PLC045956

PATIENT NAME: TUTU BEHERA PATIENT ID: TUTUM15068965

ACCESSION NO: **0065VG003259** AGE: 33 Years SEX: Male ABHA NO:

DRAWN: RECEIVED: 23/07/2022 08:34 REPORTED: 25/07/2022 15:10

REFERRING DOCTOR: SELF CLIENT PATIENT ID:

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

MEDI WHEEL FULL BODY HEALTH CHECK UP BELOW 40 MALE

BLOOD COUNTS,EDTA WHOLE BLOOD				
HEMOGLOBIN	14.7		13.0 - 17.0	g/dL
METHOD: PHOTOMETRIC MEASUREMENT				
RED BLOOD CELL COUNT	5.60	High	4.5 - 5.5	mi l /μL
METHOD : COULTER PRINCIPLE				
WHITE BLOOD CELL COUNT	8.00		4.0 - 10.0	thou/µL
METHOD : COULTER PRINCIPLE				
PLATELET COUNT	213		150 - 410	thou/µL
METHOD: ELECTRONIC IMPEDENCE & MICROSCOPY				
RBC AND PLATELET INDICES				
HEMATOCRIT	43.7		40.0 - 50.0	%
METHOD: CALCULATED PARAMETER				
MEAN CORPUSCULAR VOL	78.0	Low	83.0 - 101.0	fL
METHOD: DERIVED PARAMETER FROM RBC HISTOGRAM				
MEAN CORPUSCULAR HGB.	26.3	Low	27.0 - 32.0	pg
METHOD: CALCULATED PARAMETER				
MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION METHOD: CALCULATED PARAMETER	33.8		31.5 - 34.5	g/dL
MENTZER INDEX	13.9			
RED CELL DISTRIBUTION WIDTH	12.8		11.6 - 14.0	%
METHOD: DERIVED PARAMETER FROM RBC HISTOGRAM				
MEAN PLATELET VOLUME	9.6		6.8 - 10.9	fL
METHOD: DERIVED PARAMETER FROM PLATELET HISTOGRAM				
WBC DIFFERENTIAL COUNT - NLR				
SEGMENTED NEUTROPHILS	59		40 - 80	%
METHOD: VCSN TECHNOLOGY/ MICROSCOPY				
ABSOLUTE NEUTROPHIL COUNT	4.72		2.0 - 7.0	thou/µL
METHOD: CALCULATED PARAMETER				
LYMPHOCYTES	29		20 - 40	%
METHOD: VCSN TECHNOLOGY/ MICROSCOPY				
ABSOLUTE LYMPHOCYTE COUNT	2.32		1.0 - 3.0	thou/µL
METHOD: CALCULATED PARAMETER				
NEUTROPHIL LYMPHOCYTE RATIO (NLR)	2.0			
METHOD: CALCULATED				
EOSINOPHILS	6		1.0 - 6.0	%









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Tel: 09152729959/9111591115, CIN - U74899PB1995PLC045956

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Test Report Status <u>Final</u>	Results		Biological Reference Inte	rval Units
METHOD: VCSN TECHNOLOGY/ MICROSCOPY				
ABSOLUTE EOSINOPHIL COUNT	0.48		0.02 - 0.50	thou/µL
METHOD : CALCULATED PARAMETER				 , -
MONOCYTES	6		2.0 - 10.0	%
METHOD: VCSN TECHNOLOGY/ MICROSCOPY				
ABSOLUTE MONOCYTE COUNT	0,48		0.2 - 1.0	thou/µL
METHOD: CALCULATED PARAMETER				71
BASOPHILS	0		0 - 1	%
METHOD: VCSN TECHNOLOGY/ MICROSCOPY				
ABSOLUTE BASOPHIL COUNT	0.00	Low	0.02 - 0.10	thou/µL
METHOD: CALCULATED PARAMETER				
ERYTHRO SEDIMENTATION RATE, BLOOD				
SEDIMENTATION RATE (ESR)	17	High	0 - 14	mm at 1 hr
METHOD: AUTOMATED (PHOTOMETRICAL CAPILLARY STOP	PED FLOW KINETIC ANALYSIS)			
GLUCOSE, FASTING, PLASMA				
GLUCOSE, FASTING, PLASMA	100	High	74 - 99	mg/dL
METHOD: SPECTROPHOTOMETRY HEXOKINASE				
GLYCOSYLATED HEMOGLOBIN, EDTA WHO	DLE BLOOD			
GLYCOSYLATED HEMOGLOBIN (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: ION- EXCHANGE HPLC				
MEAN PLASMA GLUCOSE	108.3		< 116.0	mg/dL
METHOD: CALCULATED PARAMETER				
Comments				

NOTE: ABNORMAL PEAK WITH RETENTION TIME OF 1.141 NOTED, SUGGESTED VARIANT HAEMOGLOBIN STUDIES.

GLUCOSE, POST-PRANDIAL, PLASMA

GLUCOSE, POST-PRANDIAL, PLASMA 72 70 - 139 mg/dL

METHOD: SPECTROPHOTOMETRY HEXOKINASE

CORONARY RISK PROFILE (LIPID PROFILE), SERUM

CHOLESTEROL **215 High** Desirable cholesterol level mg/dL

< 200

Borderline high cholesterol

200 - 239 High cholesterol

> / = 240

METHOD: SPECTROPHOTOMETRY, ENZYMATIC COLORIMETRIC - CHOLETSEROL OXIDASE, ESTERASE, PEROXIDASE



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TRIGLYCERIDES METHOD: SPECTROPHOTO	METRY, ENZYMATIC ENDPOINT WITH	160	High	Normal: < 150 Borderline high: 150 - 199 High: 200 - 499 Very High: >/= 500	mg/dL
HDL CHOLESTEROL		34	Low	Low HDL cholesterol < 40 High HDL cholesterol > / = 60	mg/dL
METHOD : SPECTROPHOTO	METRY, HOMOGENEOUS DIRECT ENZY	MATIC COLORIMETRIC			
DIRECT LDL CHOLEST	EROL	147	High	Optimal: < 100 Near optimal/above optimal: 1 129 Borderline high: 130 - 159 High: 160 - 189 Very high: > / = 190	mg/dL 00 -
METHOD : SPECTROPHOTO	METRY, HOMOGENEOUS ENZYMATIC	COLORIMETRIC		ter,g ,	
NON HDL CHOLESTER	OL	181	High	Desirable : < 130 Above Desirable : 130 -159 Borderline High : 160 - 189 High : 190 - 219 Very high : > / = 220	mg/dL
METHOD : CALCULATED PA	RAMETER			Very mgm 1 > / 220	
CHOL/HDL RATIO		6.3	High	Low Risk: 3.3 - 4.4 Average Risk: 4.5 - 7.0 Moderate Risk: 7.1 - 11.0 High Risk: > 11.0	
METHOD : CALCULATED PA	RAMETER			3	
LDL/HDL RATIO METHOD : CALCULATED PA	RAMETER	4.3	High	Desirable/Low Risk: 0.5 - 3.0 Borderline/Moderate Risk: 3.1 6.0 High Risk: > 6.0	-
VERY LOW DENSITY L		32,0	Hiah	< or = 30.0	mg/dL
METHOD : CALCULATED PA		3210		101 - 3010	mg/ ac
LIVER FUNCTION PF					
	torice, sertori	0.76		Unto 1.2	ma/dl
BILIRUBIN, TOTAL METHOD: SPECTROPHOTO	METRY, COLORIMETRIC -DIAZO METH			Upto 1.2	mg/dL
BILIRUBIN, DIRECT		0.29	High	0.0 - 0.2	mg/dL
METHOD : SPECTROPHOTO	METRY, JENDRASSIK & GROFF - DIAZ	OTIZATION			
BILIRUBIN, INDIRECT		0.47		0.1 - 1.0	mg/dL
METHOD : CALCULATED PA	RAMETER				
TOTAL PROTEIN		7.9		6.0 - 8.0	g/dL
METHOD : SPECTROPHOTO	METRY, COLORIMETRIC -BIURET, REA	GENT BLANK, SERUM BLANK			



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ALBUMIN		- -	High	3.97 - 4.94	g/dL
	METRY, BROMOCRESOL GREEN(BCG) - DY				
GLOBULIN		2.8		2.0 - 3.5	g/dL
METHOD : CALCULATED PAR					
ALBUMIN/GLOBULIN R		1.8		1.0 - 2.1	RATIO
METHOD : CALCULATED PAR		2.4			
	ANSFERASE (AST/SGOT)	34		Upto 40	U/L
	METRY, WITHOUT PYRIDOXAL PHOSPHATE	· ·			
ALANINE AMINOTRANS	, ,		Hign	Upto 41	U/L
	METRY, WITHOUT PYRIDOXAL PHOSPHATE			40 420	117
ALKALINE PHOSPHATA		62		40 - 129	U/L
	METRY, PNPP, AMP BUFFER - IFCC	1.4			117
GAMMA GLUTAMYL TRA	` '	14		< 60	U/L
	METRY, ENZYMATIC COLORIMETRIC - G-G		LIDE -		117
LACTATE DEHYDROGEI		172		< 232	U/L
	METRY, LACTATE TO PYRUVATE - UV-IFCC				
SERUM BLOOD UREA					
BLOOD UREA NITROGE		10		6 - 20	mg/dL
	METRY, UREASE -COLORIMETRIC				
CREATININE, SERUM	1				
CREATININE		1.08		0.90 - 1.30	mg/dL
	METRY, JAFFE'S ALKALINE PICRATE KINET	TC - RATE BLANKED - IFCC-ID	MS STA	ANDARIZED	
BUN/CREAT RATIO					
BUN/CREAT RATIO		9.10		8 - 15	
METHOD : CALCULATED PAR	RAMETER				
URIC ACID, SERUM					
URIC ACID		6.8		3.4 - 7.0	mg/dL
METHOD : SPECTROPHOTON	METRY, ENZYMATIC COLORIMETRIC- URIO	CASE			
TOTAL PROTEIN, SEI	RUM				
TOTAL PROTEIN		7.9		6.0 - 8.0	g/dL
METHOD : SPECTROPHOTON	METRY, COLORIMETRIC -BIURET, REAGEN	IT BLANK, SERUM BLANK			
ALBUMIN, SERUM					
ALBUMIN		5.1	High	3.97 - 4.94	g/dL
METHOD : SPECTROPHOTON	METRY, BROMOCRESOL GREEN(BCG) - DY	E BINDING			
GLOBULIN					
GLOBULIN		2,8		2.0 - 3.5	g/dL
METHOD : CALCULATED PAR	RAMETER				









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ELECTROLYTES (NA/K/CL), SERUM	400	100 115	.,,
SODIUM	138	136 - 145	mmo l /L
METHOD: ISE INDIRECT	4.10	25 54	
POTASSIUM	4.10	3.5 - 5.1	mmo l /L
METHOD: ISE INDIRECT	100	00 100	
CHLORIDE	100	98 - 106	mmo l /L
METHOD : ISE INDIRECT			
PHYSICAL EXAMINATION, URINE	DALE VELLOW		
COLOR	PALE YELLOW		
METHOD: REFLECTANCE SPECTROPHOTOMETRY	CLEAR		
APPEARANCE	CLEAR		
METHOD: REFLECTANCE SPECTROPHOTOMETRY	1 005	1 002 1 025	
SPECIFIC GRAVITY	<=1.005	1.003 - 1.035	
METHOD: REFLECTANCE SPECTROPHOTOMETRY- PKA	CHANGE OF AN IONIC POLYELECTROLYTE		
CHEMICAL EXAMINATION, URINE			
PH	6.0	4.7 - 7.5	
METHOD: REFLECTANCE SPECTROPHOTOMETRY- DOU			
PROTEIN	NOT DETECTED	NOT DETECTED	
METHOD: REFLECTANCE SPECTROPHOTOMETRY - PRO		NOT DETECTED	
GLUCOSE	NOT DETECTED	NOT DETECTED	
METHOD: REFLECTANCE SPECTROPHOTOMETRY, DOU	•		
KETONES	NOT DETECTED	NOT DETECTED	
METHOD: REFLECTANCE SPECTROPHOTOMETRY, ROTH		NOT DETECTED	
BLOOD	NOT DETECTED	NOT DETECTED	
METHOD: REFLECTANCE SPECTROPHOTOMETRY, PERC		NOT DETECTED	
BILIRUBIN METHOD - REFLECTANCE CRECTBORHOTOMETRY DIAZ	NOT DETECTED	NOT DETECTED	
METHOD: REFLECTANCE SPECTROPHOTOMETRY, DIAZ UROBILINOGEN			
	NORMAL	NORMAL	
METHOD: REFLECTANCE SPECTROPHOTOMETRY - EHR		NOT DETECTED	
NITRITE	NOT DETECTED	NOT DETECTED	
METHOD: REFLECTANCE SPECTROPHOTOMETRY, CON- LEUKOCYTE ESTERASE	NOT DETECTED	NOT DETECTED	
	NOT DETECTED	NOT DETECTED	
MICROSCOPIC EXAMINATION, URINE			
PUS CELL (WBC'S)	0-1	0-5	/HPF
EPITHELIAL CELLS	1-2	0-5	/HPF
METHOD: MICROSCOPIC EXAMINATION			
ERYTHROCYTES (RBC'S)	NOT DETECTED	NOT DETECTED	/HPF









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CASTS NOT DETECTED	
METHOD: MICROSCOPIC EXAMINATION	
CRYSTALS NOT DETECTED	
METHOD: MICROSCOPIC EXAMINATION	
BACTERIA NOT DETECTED NOT DETECTED	
METHOD: MICROSCOPIC EXAMINATION	
YEAST NOT DETECTED NOT DETECTED	

Comments

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

NOTE: KINDLY EXERT CAUTION DURING INTERPRETATION OF FINDINGS REPORTED IN URINALYSIS WHERE IN THE SAMPLE IS MORE THAN TWO HOURS OLD.

THYROID PANEL, SERUM

T3	105.0	80.0 - 200.0	ng/dL
METHOD: COMPETITIVE ELECTROCHEMILUMINESC	ENCE IMMUNOASSAY		
T4	7.48	5.10 - 14.10	μg/dL
METHOD: COMPETITIVE ELECTROCHEMILUMINESC	ENCE IMMUNOASSAY		
TSH 3RD GENERATION	1.620	0.270 - 4.200	μIU/mL
METHOD : SANDWICH ELECTROCHEMILUMINESCEN	ICE IMMUNOASSAY		

STOOL: OVA & PARASITE			
COLOUR	BROWN		
CONSISTENCY	SEMI FORMED		
ODOUR	FAECAL		
MUCUS	NOT DETECTED	NOT DETECTED	
VISIBLE BLOOD	ABSENT	ABSENT	
POLYMORPHONUCLEAR LEUKOCYTES	0 - 1/10	0 - 5	/HPF
METHOD: MICROSCOPIC EXAMINATION			
RED BLOOD CELLS	NOT DETECTED	NOT DETECTED	/HPF
METHOD: MICROSCOPIC EXAMINATION			
MACROPHAGES	NOT DETECTED	NOT DETECTED	
METHOD: MICROSCOPIC EXAMINATION			
CHARCOT-LEYDEN CRYSTALS	NOT DETECTED	NOT DETECTED	
METHOD: MICROSCOPIC EXAMINATION			
TROPHOZOITES	NOT DETECTED	NOT DETECTED	
METHOD: MICROSCOPIC EXAMINATION			
CYSTS	NOT DETECTED	NOT DETECTED	
METHOD: MICROSCOPIC EXAMINATION			









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0.74	NOT DETECTED	
OVA METHOD: MICROSCOPIC EXAMINATION	NOT DETECTED	
LARVAE	NOT DETECTED	NOT DETECTED
METHOD: MICROSCOPIC EXAMINATION	NOT DETECTED	NOT DETECTED
ADULT PARASITE	NOT DETECTED	
METHOD: MICROSCOPIC EXAMINATION		
OCCULT BLOOD	NOT DETECTED	NOT DETECTED
METHOD: MODIFIED GUAIAC METHOD		
ABO GROUP & RH TYPE, EDTA WHOLE BLOOD		
ABO GROUP	Α	
METHOD: HAEMAGGLUTINATION (AUTOMATED)		
RH TYPE	POSITIVE	
METHOD: HAEMAGGLUTINATION (AUTOMATED)		
XRAY-CHEST		
IMPRESSION	NO ABNORMALITY DETECT	ED
TMT OR ECHO		
TMT OR ECHO	NEGATIVE	
ECG		

ECG ST & T ABNORMALITY

MEDICAL HISTORY

RELEVANT PRESENT HISTORY CVS 2 ND DOSE DONE RELEVANT PAST HISTORY DENGUE 2015 + HOSP

RELEVANT PERSONAL HISTORY ALCOHOL OCC RELEVANT FAMILY HISTORY **DIABETES**

HISTORY OF MEDICATIONS **NOT SIGNIFICANT**

ANTHROPOMETRIC DATA & BMI

HEIGHT IN METERS 1.78 mts WEIGHT IN KGS. 85 Kgs

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

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

GENERAL EXAMINATION

MENTAL / EMOTIONAL STATE **NORMAL** PHYSICAL ATTITUDE **NORMAL** GENERAL APPEARANCE / NUTRITIONAL STATUS **HEALTHY**



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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 73/MIN, REGULAR, ALL PERIPHERAL PULSES WELL FELT, NO CAROTID

BRUIT

RESPIRATORY RATE NORMAL

CARDIOVASCULAR SYSTEM

BP 122/75 MM HG mm/Hg

(SUPINE) NORMAL NORMAL

HEART SOUNDS S1, S2 HEARD NORMALLY

MURMURS ABSENT

RESPIRATORY SYSTEM

SIZE AND SHAPE OF CHEST

MOVEMENTS OF CHEST

BREATH SOUNDS INTENSITY

NORMAL

BREATH SOUNDS QUALITY VESICULAR (NORMAL)

ADDED SOUNDS ABSENT

PER ABDOMEN

PERICARDIUM

APEX BEAT

APPEARANCE NORMAL
VENOUS PROMINENCE ABSENT

LIVER NOT PALPABLE
SPLEEN NOT PALPABLE
HERNIA ABSENT

CENTRAL NERVOUS SYSTEM

HIGHER FUNCTIONS NORMAL CRANIAL NERVES NORMAL



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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(6/6	5)	
DISTANT VISION LEFT EYE WITHOUT GLASSES	WITHIN NORMAL LIMIT(6/6	5)	
NEAR VISION RIGHT EYE WITHOUT GLASSES	WITHIN NORMAL LIMIT(N/	5)	
NEAR VISION LEFT EYE WITHOUT GLASSES	WITHIN NORMAL LIMIT(N/	5)	
COLOUR VISION	NORMAL(17/17)		
BASIC ENT EXAMINATION			
EXTERNAL EAR CANAL	HEAVY WITHIN NORMAL LI	MIT	
TYMPANIC MEMBRANE	NORMAL		
NOSE	NO ABNORMALITY DETECTI	ĒD	
SINUSES	CLEAR		
THROAT	NO ABNORMALITY DETECTI	ĒD	
TONSILS	NOT ENLARGED		
SUMMARY			
RELEVANT HISTORY	NOT SIGNIFICANT		
RELEVANT GP EXAMINATION FINDINGS	NOT SIGNIFICANT		









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Tel: 09152729959/9111591115, CIN - U74899PB1995PLC045956

PATIENT NAME: TUTU BEHERA PATIENT ID: TUTUM15068965

0065VG003259 AGE: 33 Years ABHA NO: ACCESSION NO: SEX: Male

DRAWN: RECEIVED: 23/07/2022 08:34 REPORTED: 25/07/2022 15:10

REFERRING DOCTOR: SELF CLIENT PATIENT ID:

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

RAISED ESR(17). RELEVANT LAB INVESTIGATIONS RAISED RBCC(5.60).

RAISED FASTING BLOOD SUGAR(100).

RAISED SGPT(56).

RAISED DIRECT BILIRUBIN(0.29). RAISED CHOLESTEROL(215). RAISED TRIGLYCERIDES(160). LOW HDL CHOLESTEROL (34).

RAISED NON HDL CHOLESTEROL(181). RAISED DIRECT LDL CHOLESTEROL(147). RAISED VLDL CHOLESTEROL(32.0)

RAISED ALBUMIN (5.1)

USG: BORDERLINE FATTY LIVER. RELEVANT NON PATHOLOGY DIAGNOSTICS

ECG: ST & T ABNORMALITY

REMARKS / RECOMMENDATIONS REGULAR PHYSICAL EXERCISES / LOW CALORIC DIET.

REDUCE SUGARS, SWEETS IN DIET

REDUCE FATTY AND PROCESSED FOOD IN DIET

FOLLOW UP WITH PHYSICIAN FOR ABNORMAL LIPID PROFILE.

Interpretation(s)

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. 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, 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.

- 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"

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

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.



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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.

- 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, 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

CORONARY RISK PROFILE (LIPID PROFILE), SERUM-Serum cholesterol is a blood test that can provide valuable information for the risk of coronary artery disease This test can help determine your risk of the build up of plaques in your arteries that can lead to narrowed or blocked arteries throughout your body (atherosclerosis). High cholesterol levels usually don"t cause any signs or symptoms, so a cholesterol test is an important tool. High cholesterol levels often are a significant risk factor for heart disease and important for diagnosis of hyperlipoproteinemia, atherosclerosis, hepatic and thyroid diseases

Serum Triglyceride are a type of fat in the blood. When you eat, your body converts any calories it doesn't need into triglycerides, which are stored in fat cells. High triglyceride levels are associated with several factors, including being overweight, eating too many sweets or drinking too much alcohol, smoking, being sedentary, or having diabetes with elevated blood sugar levels. Analysis has proven useful in the diagnosis and treatment of patients with diabetes mellitus, nephrosis, liver obstruction, other diseases involving lipid metabolism, and various endocrine disorders. In conjunction with high density lipoprotein and total serum cholesterol, a triglyceride determination provides valuable information for the assessment of coronary heart disease risk. It is done in fasting state.

High-density lipoprotein (HDL) cholesterol. This is sometimes called the ""good"" cholesterol because it helps carry away LDL cholesterol, thus keeping arteries open and blood flowing more freely.HDL cholesterol is inversely related to the risk for cardiovascular disease. It increases following regular exercise, moderate alcohol consumption and with oral estrogen therapy. Decreased levels are associated with obesity, stress, cigarette smoking and diabetes mellitus.

SERUM LDL The small dense LDL test can be used to determine cardiovascular risk in individuals with metabolic syndrome or established/progressing coronary artery disease, individuals with triglyceride levels between 70 and 140 mg/dL, as well as individuals with a diet high in trans-fat or carbohydrates. Elevated sdLDL levels are associated with metabolic syndrome and an 'atherogenic lipoprotein profile', and are a strong, independent predictor of cardiovascular disease. Elevated levels of LDL arise from multiple sources. A major factor is sedentary lifestyle with a diet high in saturated fat. Insulin-resistance and pre-diabetes have also been implicated, as has genetic predisposition. Measurement of sdLDL allows the clinician to get a more comprehensive picture of lipid risk factors and tailor treatment accordingly, Reducing LDL levels will reduce the risk of CVD and MI,

Non HDL Cholesterol - Adult treatment panel ATP III suggested the addition of Non-HDL Cholesterol as an indicator of all atherogenic lipoproteins (mainly LDL and VLDL). NICE guidelines recommend Non-HDL Cholesterol measurement before initiating lipid lowering therapy. It has also been shown to be a better marker of risk in both primary and secondary prevention studies.

Recommendations:

Results of Lipids should always be interpreted in conjunction with the patient's medical history, clinical presentation and other findings.

NON FASTING LIPID PROFILE includes Total Cholesterol, HDL Cholesterol and calculated non-HDL Cholesterol. It does not include triglycerides and may be best used in patients for whom fasting is difficult.

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 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









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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 NITRÓGEN-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 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 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, traumá, 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



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

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

Т3 (ng/dL) (µg/dL) 1-3 day: 8.2 - 19.9 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.

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

- 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

STOOL: OVA & PARASITE-

Acute infective diarrhoea and gastroenteritis (diarrhoea with vomiting) are major causes of ill health and premature death in developing countries. Loss of water and electrolytes from the body can lead to severe dehydration which if untreated, can be rapidly fatal in young children, especially that are malnourished, hypoglycaemic, and

Laboratory diagnosis of parasitic infection is mainly based on microscopic examination and the gross examination of the stool specimen. Depending on the nature of the parasite, the microscopic observations include the identification of cysts, ova, trophozoites, larvae or portions of adult structure. The two classes of parasites that cause human infection are the Protozoa and Helminths. The protozoan infections include amoebiasis mainly caused by Entamoeba histolytica and giardiasis caused by Giardia lamblia. The common helminthic parasites are Trichuris trichiura, Ascaris lumbricoides, Strongyloides stercoralis, Taenia sp. etc ABO GROUP & RH TYPE, EDTA WHOLE BLOOD-

Blood group is identified by antigens and antibodies present in the blood. Antigens are protein molecules found on the surface of red blood cells. Antibodies are found in plasma. To determine blood group, red cells are mixed with different antibody solutions to give A,B,O or AB,

Disclaimer: "Please note, as the results of previous ABO and Rh group (Blood Group) for pregnant women are not available, please check with the patient records for availability of the same.

The test is performed by both forward as well as reverse grouping methods.

THIS REPORT CARRIES THE SIGNATURE OF OUR LABORATORY DIRECTOR, THIS IS AN INVIOLABLE FEATURE OF OUR LAB MANAGEMENT SOFTWARE, HOWEVER, ALL











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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 BELOW 40 MALE

ULTRASOUND ABDOMEN

ULTRASOUND ABDOMEN

BORDERLINE FATTY LIVER.

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

Dr. Kshama P. Biochemist

Dr. Sukanya Verma Consultant Microbiologist Dr. Deepak Sanghavi,M.D(Path) (Reg.no.MMC2004/03/1530) Chief Of Lab - Mumbai Reference Lab Dr. Sushant Chikane Consultant Pathologist

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- 1. It is presumed that the test sample belongs to the patient named or identified in the test requisition form.
- 2. All Tests are performed and reported as per the turnaround time stated in the SRL Directory of services (DOS).
- 3. SRL confirms that all tests have been performed or assayed with highest quality standards, clinical safety & technical integrity.
- 4. A requested test might not be performed if:
- a. Specimen received is insufficient or inappropriate specimen quality is unsatisfactory
 - b. Incorrect specimen type
- c. Request for testing is withdrawn by the ordering doctor or patient
- $\mbox{\rm d.}$ There is a discrepancy between the label on the specimen container and the name on the test requisition form

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- 6. Result delays could be because of uncontrolled circumstances, e.g., assay run failure.
- 7. Tests parameters marked by asterisks are excluded from the "scope" of NABL accredited tests. (If laboratory is accredited).
- 8. Laboratory results should be correlated with clinical information to determine Final diagnosis.
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