

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

SRL Ltd

S.K. Tower, Hari Niwas, LBS Marg

THANE, 400602

MAHARASHTRA, INDIA Tel: 9111591115, Fax: CIN - U74899PB1995PLC045956

Email: customercare.thane@srl.in

PATIENT NAME: MR. DILIP SHAMRAO SADAPHULE

PATIENT ID : FH.2798254

ACCESSION NO: 0181VK000384 AGE: 59 Years SEX: Male ABHA NO:

RECEIVED: 08/11/2022 12:01:04 REPORTED: 10/11/2022 16:14:51 DRAWN:

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 (HB)	15.0	13.0 - 17.0	g/dL
METHOD: SLS-HEMOGLOBIN DETECTION METHOD	F 0.7	45.55	417.1
RED BLOOD CELL (RBC) COUNT	5.27	4.5 - 5.5	mil/μL
METHOD: HYDRODYNAMIC FOCUSING BY DC DETECTION	6.06	4.0 40.0	H ()
WHITE BLOOD CELL (WBC) COUNT	6.36	4.0 - 10.0	thou/µL
METHOD: FLUORESCENCE FLOW CYTOMETRY	207	150 410	H
PLATELET COUNT	227	150 - 410	thou/µL
METHOD: HYDRODYNAMIC FOCUSING BY DC DETECTION  RBC AND PLATELET INDICES			
	45.0	40.0 50.0	0/
HEMATOCRIT (PCV)	45.9	40.0 - 50.0	%
METHOD: CUMULATIVE PULSE HEIGHT DETECTION METHO	87.1	92.0 101.0	fL
MEAN CORPUSCULAR VOLUME (MCV)	07.1	83.0 - 101.0	IL
METHOD: CALCULATED FROM RBC & HCT	28.5	27.0 - 32.0	ng
MEAN CORPUSCULAR HEMOGLOBIN (MCH)  METHOD: CALCULATED FROM THE RBC & HGB	20.3	27.0 - 32.0	pg
	32.7	31.5 - 34.5	g/dL
MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION (MCHC) METHOD: CALCULATED FROM THE HGB & HCT	32.7	31.3 - 34.3	g/ac
RED CELL DISTRIBUTION WIDTH (RDW)	12.7	11.6 - 14.0	%
METHOD: CALCULATED FROM RBC SIZE DISTRIBUTION CU	JRVE		
MENTZER INDEX	16.5		
MEAN PLATELET VOLUME (MPV)	10.4	6.8 - 10.9	fL
METHOD: CALCULATED FROM PLATELET COUNT & PLATELE	T HEMATOCRIT		
WBC DIFFERENTIAL COUNT			
NEUTROPHILS	68	40 - 80	%
METHOD: FLOW CYTOMETRY WITH LIGHT SCATTERING			
LYMPHOCYTES	23	20 - 40	%
METHOD: FLOW CYTOMETRY WITH LIGHT SCATTERING			
MONOCYTES	5	2 - 10	%
METHOD: FLOW CYTOMETRY WITH LIGHT SCATTERING			
EOSINOPHILS	4	1 - 6	%
METHOD: FLOW CYTOMETRY WITH LIGHT SCATTERING			
ABSOLUTE NEUTROPHIL COUNT	4.33	2.0 - 7.0	thou/µL
METHOD: FLOW CYTOMETRY WITH LIGHT SCATTERING			



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	SELI			OLICIA I MILLIA IB	
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ABSOLUTE LYMPHOCY  METHOD: FLOW CYTOMETI	TE COUNT  RY WITH LIGHT SCATTERING	1.45		1.0 - 3.0	thou/μL
ABSOLUTE MONOCYTE		0.29		0.2 - 1.0	thou/µL
METHOD : FLOW CYTOMET	RY WITH LIGHT SCATTERING				
ABSOLUTE EOSINOPH	IL COUNT	0.28		0.02 - 0.50	thou/µL
METHOD : FLOW CYTOMET	RY WITH LIGHT SCATTERING				
NEUTROPHIL LYMPHO	CYTE RATIO (NLR)	3.0			
MORPHOLOGY					
RBC		NORMOCYTIC NO	ORMOCHRO	DMIC	
WBC		NORMAL MORPH	OLOGY		
METHOD : MICROSCOPIC E	XAMINATION				
PLATELETS		ADEQUATE			
ERYTHROCYTE SEDI BLOOD	MENTATION RATE (E	SR),WHOLE			
E.S.R		6		< 15	mm at 1 hr
GLUCOSE FASTING,	FLUORIDE PLASMA				
FBS (FASTING BLOOD	SUGAR)	106	High	Normal 75 - 99 Pre-diabetics: 100 - 125 Diabetic: > or = 126	mg/dL
	ERENCE METHOD WITH HEXOKI				
GLYCOSYLATED HEN BLOOD	40GLOBIN(HBA1C), E	DTA WHOLE			
HBA1C		6.1	High	Non-diabetic Adult < 5.7 Pre-diabetes 5.7 - 6.4 Diabetes diagnosis: > or = 6.5 Therapeutic goals: < 7.0 Action suggested: > 8.0 (ADA Guideline 2021)	%
METHOD : HPLC	QUUQQQE(F • Q)	400.4	115-6		
ESTIMATED AVERAGE METHOD: CALCULATED PA	, ,	128.4	High	< 116.0	mg/dL
CORONARY RISK PR	OFILE, SERUM				
CHOLESTEROL, TOTAL	-	199		Desirable cholesterol level < 200 Borderline high cholesterol 200 - 239 High cholesterol > / = 240	mg/dL
METHOD - ENTIRE COL	ODIMETRIC ACCAN				

METHOD: ENZYMATIC COLORIMETRIC ASSAY







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TRIGLYCERIDES	126		Normal: < 150 Borderline high: 150 - 199 High: 200 - 499 Very High: >/= 500	mg/dL
METHOD : ENZYMATIC COLORIMETRIC ASSAY HDL CHOLESTEROL	42		Low HDL Cholesterol <40	mg/dL
METHOD : ENZYMATIC, COLORIMETRIC	_		High HDL Cholesterol >/= 60	
CHOLESTEROL LDL	132	High	Adult levels: Optimal < 100 Near optimal/above optimal: 129 Borderline high: 130-159 High: 160-189 Very high: = 190	mg/dL 100-
METHOD: ENZYMATIC COLORIMETRIC ASSAY  NON HDL CHOLESTEROL	157	High	Desirable : < 130 Above Desirable : 130 - 159 Borderline High : 160 - 189 High : 190 - 219 Very high : > / = 220	mg/dL
CHOL/HDL RATIO	4.7	High	Low Risk: 3.3 - 4.4  Average Risk: 4.5 - 7.0  Moderate Risk: 7.1 - 11.0  High Risk: > 11.0	
LDL/HDL RATIO	3.1	High	0.5 - 3.0 Desirable/Low Risk 3.1 - 6.0 Borderline/Moderate >6.0 High Risk	Risk
VERY LOW DENSITY LIPOPROTEIN	25.2		< OR = 30.0	mg/dL
LIVER FUNCTION PROFILE, SERUM				
BILIRUBIN, TOTAL  METHOD: COLORIMETRIC DIAZO	0.60		Upto 1.2	mg/dL
BILIRUBIN, DIRECT	0.26		< 0.30	mg/dL
BILIRUBIN, INDIRECT	0.34		0.1 - 1.0	mg/dL
TOTAL PROTEIN  METHOD: COLORIMETRIC	7.5		6.0 - 8.0	g/dL
ALBUMIN METHOD: COLORIMETRIC	4.8		3.97 - 4.94	g/dL
GLOBULIN	2.7		2.0 - 3.5	g/dL
ALBUMIN/GLOBULIN RATIO	1.8		1.0 - 2.1	RATIO



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ASPARTATE AMINOTRANSFERASE (AST/SGOT)	22		< OR = 50	U/L
METHOD: UV ABSORBANCE				
ALANINE AMINOTRANSFERASE (ALT/SGPT)	29		< OR = 50	U/L
METHOD: UV ABSORBANCE				_
ALKALINE PHOSPHATASE	74		40 - 129	U/L
METHOD: COLORIMETRIC	47		0. 50	
GAMMA GLUTAMYL TRANSFERASE (GGT)	17		0 - 60	U/L
METHOD: ENZYMATIC, COLORIMETRIC	222	11: -1-	105 000	1171
LACTATE DEHYDROGENASE	223	High	125 - 220	U/L
METHOD: UV ABSORBANCE				
BLOOD UREA NITROGEN (BUN), SERUM			6 00	7.11
BLOOD UREA NITROGEN	8		6 - 20	mg/dL
METHOD : ENZYMATIC ASSAY				
CREATININE, SERUM	0.75		0.7. 4.0	7.11
CREATININE	0.76		0.7 - 1.2	mg/dL
METHOD : COLORIMETRIC				
BUN/CREAT RATIO				
BUN/CREAT RATIO	10.53		8.0 - 15.0	
URIC ACID, SERUM				
URIC ACID	6.2		3.4 - 7.0	mg/dL
METHOD : ENZYMATIC COLORIMETRIC ASSAY				
TOTAL PROTEIN, SERUM				
TOTAL PROTEIN	7.5		6.0 - 8.0	g/dL
METHOD : COLORIMETRIC				
ALBUMIN, SERUM				
ALBUMIN	4.8		3.97 - 4.94	g/dL
METHOD : COLORIMETRIC				
GLOBULIN				
GLOBULIN	2.7		2.0 - 3.5	g/dL
ELECTROLYTES (NA/K/CL), SERUM				
SODIUM, SERUM	140		136 - 145	mmol/L
POTASSIUM, SERUM	4.26		3.5 - 5.1	mmol/L
CHLORIDE, SERUM	99		98 - 107	mmol/L
Interpretation(s)				



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PHYSICAL EXAMINATION	ON, URINE				
COLOR		PALE YELLOW			
APPEARANCE		CLEAR			
CHEMICAL EXAMINATI	ON, URINE				
PH		6.0		5.00 - 7.50	
SPECIFIC GRAVITY		1.005	Low	1.010 - 1.030	
PROTEIN		NOT DETECTED		NOT DETECTED	
GLUCOSE		NOT DETECTED		NOT DETECTED	
KETONES		NOT DETECTED		NOT DETECTED	
BLOOD		NOT DETECTED		NOT DETECTED	
UROBILINOGEN		NORMAL		NORMAL	
NITRITE		NOT DETECTED		NOT DETECTED	
LEUKOCYTE ESTERASE		NOT DETECTED		NOT DETECTED	
MICROSCOPIC EXAMIN	NATION, URINE				
RED BLOOD CELLS		NOT DETECTED		NOT DETECTED	/HPF
PUS CELL (WBC'S)		1-2		0-5	/HPF
EPITHELIAL CELLS		1-2		0-5	/HPF
CASTS		NOT DETECTED			
CRYSTALS		NOT DETECTED			
BACTERIA		NOT DETECTED		NOT DETECTED	
YEAST		NOT DETECTED		NOT DETECTED	
Interpretation(s)					
THYROID PANEL, SERU	IM				
T3	) ri	136.0		80 - 200	ng/dL
METHOD : ELECTROCHEMILUMI	INESCENCE	130.0		80 - 200	пулас
T4		9.35		5.1 - 14.1	μg/dL
METHOD : ELECTROCHEMILUMI	INESCENCE				. <i>31</i>
TSH 3RD GENERATION		3.180		0.27 - 4.2	μIU/mL
METHOD : ELECTROCHEMILUMI	INESCENCE				



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## Interpretation(s)

Triiodothyronine T3, Thyroxine T4, and Thyroid Stimulating Hormone TSH are thyroid hormones which affect 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.

Excessive secretion of thyroxine in the body is hyperthyroidism, and deficient secretion is called hypothyroidism.

In primary hypothyroidism, TSII levels are significantly clevated, while in secondary and tertiary hyporthyroidism, TSII levels are low. owidetlparowidetlparBelow mentioned are the guidelines for Pregnancy related reference ranges for Total T4, TSII & Total T3. Measurement of the serum TT3 level is a more sensitive test for the diagnosis of hypothyroidism, and measurement of TT4 is more useful in the diagnosis of 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. It is advisable to detect Free T3, FreeT4 along with TSH, instead of testing for albumin bound Total T3, Total T4.

Sr. No.	TSH	Total T4	FT4	Total T3	Possible Conditions
1	High	Low	Low	Low	(1) Primary Hypothyroidism (2) Chronic autoimmune Thyroiditis (3) Post Thyroidectomy (4) Post Radio-Iodine treatment
2	High	Normal	Normal	Normal	(1)Subclinical Hypothyroidism (2) Patient with insufficient thyroid hormone replacement therapy (3) In cases of Autoimmune/Hashimoto thyroiditis (4). Isolated increase in TSH levels can be due to Subclinical inflammation, drugs like amphetamines, lodine containing drug and dopamine antagonist e.g. domperidone and other physiological reasons.
3	Normal/Low	Low	Low	Low	(1) Secondary and Tertiary Hypothyroidism
4	Low	High	High	High	(1) Primary Hyperthyroidism (Graves Disease) (2) Multinodular Goitre (3)Toxic Nodular Goitre (4) Thyroiditis (5) Over treatment of thyroid hormone (6) Drug effect e.g. Glucocorticoids, dopamine, T4 replacement therapy (7) First trimester of Pregnancy
5	Low	Normal	Normal	Normal	(1) Subclinical Hyperthyroidism
6	High	High	High	High	(1) TSH secreting pituitary adenoma (2) TRH secreting tumor
7	Low	Low	Low	Low	(1) Central Hypothyroidism (2) Euthyroid sick syndrome (3) Recent treatment for Hyporthyroidism
8	Normal/Low	Normal	Normal	High	(1) T3 thyrotoxicosis (2) Non-Thyroidal illness
9	Low	High	High	Normal	(1) T4 Ingestion (2) Thyroiditis (3) Interfering Anti TPO antibodies

REF: 1. TIETZ Fundamentals of Clinical chemistry 2.Guidlines of the American Thyroid association during pregnancy and Postpartum, 2011. NOTE: It is advisable to detect Free T3,FreeT4 along with TSH, instead of testing for albumin bound Total T3, Total T4.TSH is not affected by variation in thyroid - binding protein. TSH has a diurnal rhythm, with peaks at 2:00 - 4:00 a.m. And troughs at 5:00 - 6:00 p.m. With ultradian variations.

## STOOL: OVA & PARASITE

COLOUR BROWN

METHOD : VISUAL

CONSISTENCY SEMI FORMED

METHOD : VISUAL



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ODOUR	FAECAL		
METHOD : PHYSICAL			
MUCUS	ABSENT	NOT DETECTED	
METHOD: VISUAL			
VISIBLE BLOOD	ABSENT	ABSENT	
METHOD: VISUAL			
POLYMORPHONUCLEAR LEUKOCYTES	2-3	0 - 5	/HPF
METHOD: MICROSCOPIC EXAMINATION			
RED BLOOD CELLS	NOT DETECTED	NOT DETECTED	/HPF
METHOD: MICROSCOPIC EXAMINATION			
TROPHOZOITES	NOT DETECTED	NOT DETECTED	
METHOD: MICROSCOPIC EXAMINATION			
CYSTS	NOT DETECTED	NOT DETECTED	
METHOD: MICROSCOPIC EXAMINATION			
OVA	NOT DETECTED		
METHOD: MICROSCOPIC EXAMINATION			
LARVAE	NOT DETECTED	NOT DETECTED	
METHOD: MICROSCOPIC EXAMINATION			
OCCULT BLOOD	NOT DETECTED	NOT DETECTED	
METHOD: HEMOSPOT			
REMARK	NO OVA CYST SEEN AFTER FOR STOOL SAMPLE.	R PERFORMING CONCENTRATION	TECHNIQUE
Interpretation(s)			

# 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 :-

Post ASD-ICR, No residual shunt. Aortic valve sclerosis, Mild AR

Mild Concentric LVH.

**ECG** 







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ECG T ABNORMALITY IN ANTEROLATERAL LEADS.

MEDICAL HISTORY

RELEVANT PRESENT HISTORY C/O DYSPNOEA ON EXERTION.

RETINAL SURGERY DONE 4 YEARS BACK. LEFT TYMPANOPLASTY 15 YEARS BACK.

SEPTOPLASTY 5 YEARS BACK.

OPEREAED FOR BILATERAL CATARACT 8 YEARS BACK.

RELEVANT PAST HISTORY PAST H/O PROSTATOMEGALY -TAKEN TREATMENT NO DETAILS.

OPERSTED ASD REPAIR IN 1979 OPEREAED FOR PILES 6 YEARS BACK MALARIA DENGUE SEVERAL TIMS

COVID 2 EARS BACK.

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

OCC. ALCOHOL.

RELEVANT FAMILY HISTORY FATHER;- TUBERULOSIS.
HISTORY OF MEDICATIONS SAMPLE NOT RECEIVED

ANTHROPOMETRIC DATA & BMI

HEIGHT IN METERS 1.65 mts WEIGHT IN KGS. 78 Kgs

BMI & Weight Status as follows: kg/sqmts

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

**GENERAL EXAMINATION** 

MENTAL / EMOTIONAL STATE NORMAL PHYSICAL ATTITUDE NORMAL GENERAL APPEARANCE / NUTRITIONAL STATUS **OVERWEIGHT BUILT / SKELETAL FRAMEWORK AVERAGE** FACIAL APPEARANCE NORMAL SKIN NORMAL UPPER LIMB **NORMAL** LOWER LIMB NORMAL NECK NORMAL

NECK LYMPHATICS / SALIVARY GLANDS NOT ENLARGED OR TENDER

THYROID GLAND NOT ENLARGED

CAROTID PULSATION NORMAL



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TEMPERATURE	NORMAL		
PULSE	68/MIN.REGULAR, AL BRUIT	L PERIPHERAL PULSES WELL FELT, NO CAROTID	
RESPIRATORY RATE	NORMAL		
CARDIOVASCULAR SYSTEM			
ВР	160/90 MM HG (SUPINE)	mm/Hg	
PERICARDIUM	NORMAL		
APEX BEAT	NORMAL		
HEART SOUNDS	NORMAL		
MURMURS	ABSENT		
RESPIRATORY SYSTEM			
SIZE AND SHAPE OF CHEST	NORMAL		
MOVEMENTS OF CHEST	SYMMETRICAL		
BREATH SOUNDS INTENSITY	NORMAL		
BREATH SOUNDS QUALITY	VESICULAR (NORMAL	_)	
ADDED SOUNDS	ABSENT		
PER ABDOMEN			
APPEARANCE	NORMAL		

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** 







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PATIENT NAME: MR. DILIP SHAMRAO SADAPHULE

PATIENT ID : FH.2798254

ACCESSION NO: 0181VK000384 AGE: 59 Years SEX: Male ABHA NO:

RECEIVED: 08/11/2022 12:01:04 REPORTED: 10/11/2022 16:14:51 DRAWN:

REFERRING DOCTOR: SELF CLIENT PATIENT ID:

Test Report Status <u>Final</u>	Results	Biological Reference Interval Units
CONJUNCTIVA	NORMAL	
EYELIDS	NORMAL	
EYE MOVEMENTS	NORMAL	
CORNEA	NORMAL	
DISTANT VISION RIGHT EYE WITHOUT GLASSES	REDUCED VISUAL ACUITY	6/18
DISTANT VISION LEFT EYE WITHOUT GLASSES	REDUCED VISUAL ACUITY	6/9
NEAR VISION RIGHT EYE WITHOUT GLASSES	WITHIN NORMAL LIMIT	
NEAR VISION LEFT EYE WITHOUT GLASSES	WITHIN NORMAL LIMIT	
COLOUR VISION	NORMAL	
SUMMARY		
RELEVANT HISTORY	NOT SIGNIFICANT	
RELEVANT GP EXAMINATION FINDINGS	OVERWEIGHT :- BMI 29	
REMARKS / RECOMMENDATIONS	3)OPHTHALMOLOGY CONS	R CONTROL OF BP. LT IN VIEW OF ECG FINDINGS. SULT FOR REDUCED VISUAL ACUITY , LOW CARBOHYDRATE, HIGH FIBRE DIET.
	5)REGULAR EXERCISE.REG	GULAR WALK FOR 30-40 MIN DAILY.
	6)REPEAT LIPID PROFILE, AND EXERCISE. 7)TO DO PSA. 8)SURGICAL CONSULT FO	BLOOD SUGAR AFTER 3 MONTHS OF DIET R UMBILICAL HERNIA.







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### MEDI WHEEL FULL BODY HEALTH CHECK UP BELOW 40 MALE

**ULTRASOUND ABDOMEN** 

**ULTRASOUND ABDOMEN** 

GRADE I FATTY LIVER MILD PROSTATOMEGALY SMALL UMBILICAL HERNIA

## Interpretation(s)

BLOOD COUNTS, ED TA 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 dinical correlation and suspicion. Estimation of HbA2 remains the gold standard for diagnosing a case of beta thalassaemia trait. WBC DIFFERENTIAL COUNT-

The optimal threshold of 3.3 for NLR showed a prognostic possibility of dinical 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.
ERYTHROCYTE SEDIMENTATION RATE (ESR), WHOLE BLOOD-TEST DESCRIPTION:

(sedimentation) of erythrocytes in a sample of blood that has been placed into a tall, thin, vertical tube. Results are reported as the millimetres of dear fluid (plasma) that are present at the top portion of the tube after one hour. Nowadays fully automated instruments are available to measure ESR.

ESR is not diagnostic; it is a non-specific test that may be elevated in a number of different conditions. It provides general information about the presence of an inflammatory condition.CRP is superior to ESR because it is more sensitive and reflects a more rapid change. **TEST INTERPRETATION** 

Increase in: Infections, Vasculities, Inflammatory arthritis, Renal disease, Anemia, Malignancies and plasma cell dyscrasias, Acute allergy Tissue injury, Pregnancy, Estrogen medication, Aging.
Finding a very accelerated ESR(>100 mm/hour) in patients with ill-defined symptoms directs the physician to search for a systemic disease (Paraproteinemias,

Disseminated malignancies, connective tissue diséase, severe infections such as bacterial endocarditis).
In pregnancy BRI in first trimester is 0-48 mm/hr(62 if anemic) and in second trimester (0-70 mm/hr(95 if anemic). ESR returns to normal 4th week post partum.

Decreased in: Polycythermia vera, Sickle cell anemia

False elevated ESR: Increased fibrinogen, Drugs(Vitamin A, Dextran etc), Hypercholesterolemia
False Decreased: Poikilocytosis,(SickleCells,spherocytes),Microcytosis, Low fibrinogen, Very high WBC counts, Drugs(Quinine, salicylates)

## 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.

GLUCOSE FASTING, FLUORIDE PLASMA-TEST DESCRÍPTION

Normally, the glucose concentration in extracellular fluid is closely regulated so that a source of energy is readily available to tissues and sothat no glucose is excreted in the urine.

### Increased in

Diabetes mellitus, Cushing's syndrome (10 – 15%), chronic pancreatitis (30%). Drugs:corticosteroids,phenytoin, estrogen, thiazides.

Pancreatic islet cell disease with increased insulin, insulinoma, adrenocortical insufficiency, hypopituitarism, diffuse liver disease, malignancy (adrenocortical, stomach, fibrosarcoma), infant of a diabetic mother, enzyme deficiency diseases(e.g., galactosemia), Drugs-insulin, ethanol, propranolol; sulfonylureas, tolbutamide, and other oral hypoglycemic agents.

NOTE:

Hypoglycemia is defined as a glucoseof < 50 mg/dL in men and< 40 mg/dL in women.

While random serum glucose levels correlate with home glucose monitoring results (weekly mean capillary glucose values), there is wide fluctuation within individuals. Thus,







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glycosylated hemoglobin(HbA1c) levels are favored to monitor glycemic control.
High fasting glucose level in comparison to post prandial glucose level may be seen due to effect of Oral Hypoglycaemics & Insulin treatment, Renal Glyosuria, Glycaemic index & response to food consumed, Alimentary Hypoglycemia, Increased insulin response & sensitivity etc.
GLYCOSYLATED HEMOGLOBIN(HBA1C), EDTA WHOLE BLOOD-**Used For**:

1.Evaluating the long-term control of blood glucose concentrations in diabetic patients.

Diagnosing diabetes.
 Identifying patients at increased risk for diabetes (prediabetes).

The ADA recommends measurement of HbA1c (typically 3-4 times per year for type 1 and poorly controlled type 2 diabetic patients, and 2 times per year for well-controlled type 2 diabetic patients) to determine whether a patients metabolic control has remained continuously within the target range.

1.eAG (Estimated average glucose) converts percentage HbA1c to md/dl, to compare blood glucose levels.
2. eAG gives an evaluation of blood glucose levels for the last couple of months.
3. eAG is calculated as eAG (mg/dl) = 28.7 \* HbA1c - 46.7

### HbA1c Estimation can get affected due to:

I.Shortened Erythrocyte survival: Any condition that shortens erythrocyte survival or decreases mean erythrocyte age (e.g. recovery from acute blood loss, hemolytic anemia) will falsely lower HbA1c test results. Fructosamine is recommended in these patients which indicates diabetes control over 15 days.

II. Vitamin C & E are reported to falsely lower test results. (possibly by inhibiting glycation of hemoglobin.

IIII. Iron deficiency anemia is reported to increase test results. Hypertrigiveridemia, uremia, hyperbilirubinemia, chronic alcoholism, chronic ingestion of salicylates & opiates addiction are reported to interfere with some assay methods, falsely increasing results.

addiction are reported to interfere with some assay methods, falsely increasing results.

IV.Interference of hemoglobinopathies in HbA1c estimation is seen in
a.Homozygous hemoglobinopathy. Fructosamine is recommended for testing of HbA1c.
b.Heterozygous state detected (D10 is corrected for HbS & HbC trait.)
c.HbF > 25% on alternate paltform (Boronate affinity chromatography) is recommended for testing of HbA1c.Abnormal Hemoglobin electrophoresis (HPLC method) is
recommended for detecting a hemoglobinopathy
LIVER FUNCTION PROFILE, SERUMLIVER 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

may be a result of Hemolytic or perindous anemia, Transfusion reaction & a common metabolic condition termed Glibert 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 musde, kidneys, brain, and red blood cells, and it is commonly measured dinically 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, musdes, and pancreas. It is commonly measured as a part of a diagnostic evaluation of hepatics, obstruction of bile ducts, cirrhosis.

ALS is a restored from the part of all levels are soon in 9 library electrostics.

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, osteomaladia, 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:

Agamman(hyblinemia, Bleeding (hemogrhage) Burns, Glomerulpenhitis, Liver disease, Malabscrotion, Malutrition, Nenhordic syndrome, Protein-Insign enteroprative to: Human 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

BLOOD UREA NITROGEN (BUN), SERUM-Causes of Increased levels include Pre renal (High protein diet, Increased protein catabolism, GI haemorrhage, Cortisol, Dehydration, CHF Renal), Renal Failure, Post Renal (Malignancy, Nephrolithiasis, Prostatism)
Causes of decreased level include 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)
   Musde problems, such as breakdown of muscle fibers
- Problems during pregnancy, such as seizures (eclampsia)), or high blood pressure caused by pregnancy (preedampsia)

Lower than normal level may be due to:

Myasthenia Gravis







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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, Multiple Sclerosis 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. 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 dearance, malnutrition and wasting etc.

ABO GROUP & RH TYPE, EDTA WHOLE BLOODBlood 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 EXAMINATIONS AND INVESTIGATIONS HAVE BEEN CONDUCTED BY OUR PANEL OF DOCTORS.

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



