

CODE/NAME & ADDRESS: C000138363

ARCOFEMI HEALTHCARE LTD (MEDIWHEEL
F-703, LADO SARAI, MEHRAULISOUTH WEST

DELHI

NEW DELHI 110030 8800465156 ACCESSION NO: **0031XC020455**PATIENT ID : TANIF22098231

CHIENT BATIENT ID:

AGE/SEX :41 Years Female DRAWN :22/03/2024 10:22:00

RECEIVED : 22/03/2024 10:25:04 REPORTED :30/03/2024 20:12:10

Test Report Status Final Results Biological Reference Interval Units

MEDI WHEEL FULL BODY HEALTH CHECKUP ABOVE 40FEMALE

XRAY-CHEST

IMPRESSION NO ABNORMALITY DETECTED

ECG

ECG NORMAL

MAMOGRAPHY (BOTH BREASTS)

MAMOGRAPHY BOTH BREASTS TEST PENDING

MEDICAL HISTORY

RELEVANT PRESENT HISTORY MYOLIPOMA OF ADRENAL GLAND

RELEVANT PAST HISTORY COVID

RELEVANT PERSONAL HISTORY NOT SIGNIFICANT

RELEVANT FAMILY HISTORY PARENTS: HTN, DIABETIC

OCCUPATIONAL HISTORY NOT SIGNIFICANT HISTORY OF MEDICATIONS NOT SIGNIFICANT

ANTHROPOMETRIC DATA & BMI

HEIGHT IN METERS 1.52 mts
WEIGHT IN KGS. 77 Kgs

BMI 33 BMI & Weight Status as follows/sqm

MI 33 BMI & Weight Status as follows/sqmts
Below 18.5: Underweight

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

Desilve Roy

Dr. Debika Roy

MBBS Consultant Physician





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GENERAL EXAMINATION

MENTAL / EMOTIONAL STATE NORMAL
PHYSICAL ATTITUDE NORMAL
GENERAL APPEARANCE / NUTRITIONAL OBESE

STATUS

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 76/MINS-REGULAR, ALL PERIPHERAL PULSES WELL FELT

RESPIRATORY RATE NORMAL

CARDIOVASCULAR SYSTEM

BP 130/86 mm/Hg

PERICARDIUM NORMAL APEX BEAT NORMAL

HEART SOUNDS S1, S2 HEARD NORMALLY

MURMURS ABSENT

RESPIRATORY SYSTEM

SIZE AND SHAPE OF CHEST NORMAL

Desilve Ray

Dr. Debika Roy

MBBS Consultant Physician





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PERFORMED AT :

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MOVEMENTS OF CHEST **SYMMETRICAL** BREATH SOUNDS INTENSITY **NORMAL**

VESICULAR (NORMAL) BREATH SOUNDS QUALITY

ADDED SOUNDS **ABSENT**

PER ABDOMEN

APPEARANCE NORMAL VENOUS PROMINENCE **ABSENT**

LIVER **NOT PALPABLE NOT PALPABLE SPLEEN HERNIA ABSENT**

CENTRAL NERVOUS SYSTEM

NORMAL HIGHER FUNCTIONS CRANIAL NERVES **NORMAL NORMAL** CEREBELLAR FUNCTIONS **NORMAL** SENSORY SYSTEM **NORMAL** MOTOR SYSTEM **NORMAL** REFLEXES

MUSCULOSKELETAL SYSTEM

SPINE **NORMAL JOINTS NORMAL**

BASIC EYE EXAMINATION

CONJUNCTIVA **NORMAL**

Desile Ray

Dr. Debika Roy **MBBS Consultant Physician**





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EYELIDS NORMAL
EYE MOVEMENTS NORMAL
DISTANT VISION RIGHT EYE WITH GLASSES 6/6
DISTANT VISION LEFT EYE WITH GLASSES 6/6
NEAR VISION RIGHT EYE WITH GLASSES N6
NEAR VISION LEFT EYE WITH GLASSES N6
COLOUR VISION NORMAL

BASIC ENT EXAMINATION

EXTERNAL EAR CANAL NORMAL TYMPANIC MEMBRANE NORMAL

NOSE NO ABNORMALITY DETECTED

SINUSES CLEAR

THROAT NO ABNORMALITY DETECTED

TONSILS NOT ENLARGED

BASIC DENTAL EXAMINATION

TEETH NORMAL GUMS HEALTHY

SUMMARY

RELEVANT HISTORY MYOLIPOMA OF ADRENAL GLAND

RELEVANT GP EXAMINATION FINDINGS OBESE (77 KGS)

RELEVANT LAB INVESTIGATIONS RAISED LDL (114), NON HDL (144)
RELEVANT NON PATHOLOGY DIAGNOSTICS NO ABNORMALITIES DETECTED

Desilve Roy

Dr. Debika Roy MBBS Consultant Physician



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REMARKS / RECOMMENDATIONS

On examination and investigations the candidate is found to be obese and has raised LDL (114), NON HDL (144)

Should follow the given advice:

- 1. Avoid fat and oily diet
- 2. Reduce body weight
- 3. Estimated body weight should be : 55 kg4. Regular physical exercise and walking
- 5. Drink plenty of water
- 6. Physician and Dietician consultation

Comments

MEDICAL EXAMINATION DONE BY:

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

Desilve Ray

Dr. Debika Roy MBBS Consultant Physician

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MEDI WHEEL FULL BODY HEALTH CHECKUP ABOVE 40FEMALE

ULTRASOUND ABDOMEN

ULTRASOUND ABDOMEN

TEST PENDING

TMT OR ECHO
CLINICAL PROFILE

TMT DONE- NEGATIVE

Interpretation(s)

MEDICAL

THIS REPORT CARRIES THE SIGNATURE OF OUR LABORATORY DIRECTOR. THIS IS AN INVIOLABLE FEATURE OF OUR LAB MANAGEMENT SOFTWARE. HOWEVER, ALL EXAMINATIONS AND INVESTIGATIONS HAVE BEEN CONDUCTED BY OUR PANEL OF DOCTORS.

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н	IAEMATOLOGY - CBC	2	
MEDI WHEEL FULL BODY HEALTH CHECKUP A	BOVE 40FEMALE		
BLOOD COUNTS,EDTA WHOLE BLOOD			
HEMOGLOBIN (HB) METHOD: SPECTROPHOTOMETRY	12.0	12.0 - 15.0	g/dL
RED BLOOD CELL (RBC) COUNT METHOD: ELECTRICAL IMPEDANCE	4.58	3.8 - 4.8	mil/μL
WHITE BLOOD CELL (WBC) COUNT METHOD: ELECTRICAL IMPEDANCE	7.58	4.0 - 10.0	thou/μL
PLATELET COUNT METHOD: ELECTRONIC IMPEDENCE & MICROSCOPY	253	150 - 410	thou/μL
RBC AND PLATELET INDICES			
HEMATOCRIT (PCV) METHOD: CALCULATED	36.8	36 - 46	%
MEAN CORPUSCULAR VOLUME (MCV) METHOD: ELECTRICAL IMPEDANCE	80.3 Low	83 - 101	fL
MEAN CORPUSCULAR HEMOGLOBIN (MCH) METHOD: CALCULATED	26.3 Low	27.0 - 32.0	pg
MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION (MCHC) METHOD: CALCULATED	32.7	31.5 - 34.5	g/dL
RED CELL DISTRIBUTION WIDTH (RDW) METHOD: ELECTRICAL IMPEDANCE	15.2 High	11.6 - 14.0	%
MENTZER INDEX	17.5		
MEAN PLATELET VOLUME (MPV) METHOD: CALCULATED	9.4	6.8 - 10.9	fL
WBC DIFFERENTIAL COUNT			
NEUTROPHILS	60	40 - 80	%
METHOD: FLOWCYTOMETRY, ELECTRONIC IMPEDANCE & MICROSOL LYMPHOCYTES	COPY. 30	20 - 40	%
METHOD: FLOWCYTOMETRY, ELECTRONIC IMPEDANCE & MICROS	COPY.		

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	<u> </u>		
Test Report Status <u>Final</u>	Results	Biological Reference	e Interval Units
MONOCYTES	7	2 - 10	%
METHOD: FLOWCYTOMETRY, ELECTRONIC IMPEDANCE & I	MICROSCOPY.		
EOSINOPHILS	3	1 - 6	%
BASOPHILS	0	0 - 2	%
METHOD: FLOWCYTOMETRY, ELECTRONIC IMPEDANCE & I	MICROSCOPY.		
ABSOLUTE NEUTROPHIL COUNT	4.55	2.0 - 7.0	thou/µL
METHOD: FLOWCYTOMETRY & CALCULATED			
ABSOLUTE LYMPHOCYTE COUNT	2.27	1 - 3	thou/μL
METHOD: FLOWCYTOMETRY & CALCULATED			
ABSOLUTE MONOCYTE COUNT	0.53	0.20 - 1.00	thou/μL
METHOD: FLOWCYTOMETRY & CALCULATED			
ABSOLUTE EOSINOPHIL COUNT	0.23	0.02 - 0.50	thou/μL
METHOD: FLOWCYTOMETRY & CALCULATED			
ABSOLUTE BASOPHIL COUNT	0.00 Low	0.02 - 0.10	thou/μL
METHOD: FLOWCYTOMETRY & CALCULATED			

MORPHOLOGY

PREDOMINANTLY NORMOCYTIC NORMOCHROMIC RBC

METHOD: MICROSCOPIC EXAMINATION NO IMMATURE CELLS SEEN. **WBC**

METHOD: MICROSCOPIC EXAMINATION

ADEQUATE PLATELETS

METHOD: MICROSCOPIC EXAMINATION

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.

was considered that seems to be the control of the 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.

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HAEMATOLOGY

MEDI WHEEL FULL BODY HEALTH CHECKUP ABOVE 40FEMALE

ERYTHROCYTE SEDIMENTATION RATE (ESR), EDTA BLOOD

E.S.R 12 0 - 20 mm at 1 hr

METHOD: AUTOMATED (PHOTOMETRICAL CAPILLARY STOPPED FLOW KINETIC ANALYSIS)"

GLYCOSYLATED HEMOGLOBIN(HBA1C), EDTA WHOLE BLOOD

HBA1C 5.5 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

ESTIMATED AVERAGE GLUCOSE(EAG) 111.2 < 116.0 mg/dL

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AGILUS DIAGNOSTICS LIMITED - KOLKATA Bio-Rad Variant II Turbo CDM 5.4 S/N: 13466

PATIENT REP V2TURBO_A1c

22/03/2024 13:46:30

3160

201

Patient Data

Sample ID: Patient ID: Name: Physician: Sex: DOB: 3107502498

Analysis Data
Analysis Performed:
Injection Number:
Run Number:
Rack ID:

Rack ID:
Tube Number: 10

Report Generated: 22/03/2024 14:06:26

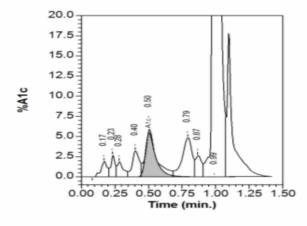
Operator ID:

Comments:

Peak Name	NGSP %	Area %	Retention Time (min)	Peak Area
A1a		1.0	0.167	19469
A1b		0.9	0.231	17730
F		1.0	0.280	18387
LA1c		1.9	0.401	37124
A1c	5.5		0.503	88974
P3		4.0	0.790	75599
P4		1.3	0.866	25182
Ao		85.2	0.990	1625804

Total Area: 1,908,268

HbA1c (NGSP) = 5.5 %



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REF. DOCTOR: DR. ARCOFEMI HEALTHCARE LTD **PATIENT NAME: TANIMA LAMA** (MEDIWHEEL

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Interpretation(s)

ERYTHROCYTE SEDIMENTATION RATE (ESR), EDTA BLOOD-**TEST DESCRIPTION**:Erythrocyte sedimentation rate (ESR) is a test that indirectly measures the degree of inflammation present in the body. The test actually measures the rate of fall (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 clear 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 disease, 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

LIMITATIONS

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)

- 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(HBA1C), EDTA WHOLE BLOOD-**Used For**:
- 1. Evaluating the long-term control of blood glucose concentrations in diabetic patients.
- 2. Diagnosing diabetes.3. 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 :

- 1. 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. 2. Vitamin C & E are reported to falsely lower test results. (possibly by inhibiting glycation of hemoglobin.
- 3. Iron deficiency anemia is reported to increase test results. Hypertriglyceridemia, uremia, hyperbilirubinemia, chronic alcoholism, chronic ingestion of salicylates & opiates addiction are reported to interfere with some assay methods, falsely increasing results.
- 4. 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

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IMMUNOHAEMATOLOGY

MEDI WHEEL FULL BODY HEALTH CHECKUP ABOVE 40FEMALE

ABO GROUP & RH TYPE, EDTA WHOLE BLOOD

ABO GROUP TYPE O

METHOD: GEL CARD METHOD

RH TYPE **POSITIVE**

METHOD : GEL CARD METHOD

Interpretation(s)

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.

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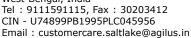


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West Bengal, India Tel: 9111591115, Fax: 30203412 CIN - U74899PB1995PLC045956









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BIOCHEMISTRY

90

MEDI WHEEL FULL BODY HEALTH CHECKUP ABOVE 40FEMALE

GLUCOSE FASTING, FLUORIDE PLASMA

FBS (FASTING BLOOD SUGAR)

METHOD: ENZYMATIC (HEXOKINASE/G-6-PDH)

mg/dL

GLUCOSE, POST-PRANDIAL, PLASMA

PPBS(POST PRANDIAL BLOOD SUGAR)

TEST NOT

140 Normal

74 - 100

mg/dL

PERFORMED

140 - 199 Pre-diabetic

> or = 200 Diabetic

METHOD: ENZYMATIC (HEXOKINASE/G-6-PDH)

LIPID PROFILE WITH CALCULATED LDL, SERUM

CHOLESTEROL, TOTAL 195

150

< 200 Desirable

mg/dL

200 - 239 Borderline High >/= 240 High

METHOD: ENZYMATIC ASSAY

TRIGLYCERIDES

METHOD: ACCELERATOR SELECTIVE DETERGENT METHODOLOGY

< 150 Normal

mg/dL

150 - 199 Borderline High

200 - 499 High >/=500 Very High

METHOD: GLYCEROL PHOSPHATE OXIDASE

HDL CHOLESTEROL

CHOLESTEROL LDL

51

114 High

Low: < 40

mg/dL

High: > / = 60

Optimal : < 100 mg/dL

Near optimal/above optimal:

100-129

Borderline high: 130-159

High: 160-189

Very high: > or = 190

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Dr.Anwesha

Chatterjee, MD, DipRCPath (Histopathology) Pathologist



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Dr. Chaitali Ray, PHD

Chief Biochemist cum MRQA

Agilus Diagnostics Ltd. P S Srijan Tech Park Building, Dn-52, Unit No. 2, Ground Floor, Sector V, Salt Lake, Kolkata, 700091

West Bengal, India







CODE/NAME & ADDRESS: C000138363 ACCESSION NO: 0031XC020455 AGE/SEX: 41 Years Female

ARCOFEMI HEALTHCARE LTD (MEDIWHEEL PATIENT ID: TANIF22098231 DRAWN: 22/03/2024 10:22:00

F-703, LADO SARAI, MEHRAULISOUTH WEST

DELHI

NEW DELHI 110030

RECEIVED : 22/03/2024 10:25:04

REPORTED : 30/03/2024 20:12:10

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

NON HDL CHOLESTEROL **144 High** Desirable: Less than 130 mg/dL

Above Desirable: 130-159 Borderline High: 160-189

High: 190 -219

Very High: >or = 220

METHOD: CALCULATED

8800465156

VERY LOW DENSITY LIPOPROTEIN 30.0 mg/dL

CHOL/HDL RATIO 3.8 LDL/HDL RATIO 2.2

Interpretation(s)

Serum lipid profile is measured for cardiovascular risk prediction. Lipid Association of India recommends LDL-C as primary target and Non HDL-C as co-primary treatment target.

Risk Stratification for ASCVD (Atherosclerotic cardiovascular disease) by Lipid Association of India

Risk Category				
Extreme risk group	A.CAD with > 1 feature of high risk group			
	B. CAD with > 1 feature of Very high risk g	group or recurrent ACS (within 1 year) despite LDL-C < or =		
	50 mg/dl or polyvascular disease			
Very High Risk	1. Established ASCVD 2. Diabetes with 2 r	najor risk factors or evidence of end organ damage 3.		
	Familial Homozygous Hypercholesterolemia	a		
High Risk	1. Three major ASCVD risk factors. 2. Diabetes with 1 major risk factor or no evidence of end organ			
	damage. 3. CKD stage 3B or 4. 4. LDL >190 mg/dl 5. Extreme of a single risk factor. 6. Coronary			
	Artery Calcium - CAC >300 AU. 7. Lipoprotein a >/= 50mg/dl 8. Non stenotic carotid plaque			
Moderate Risk	2 major ASCVD risk factors			
Low Risk	0-1 major ASCVD risk factors			
Major ASCVD (Atherosclerotic cardiovascular disease) Risk Factors				
1. Age > or = 45 years in males and > or = 55 years in females 3. Current Cigarette smoking or tobacco use				
2. Family history of premature ASCVD 4. High blood pressure				
5. Low HDL				

Newer treatment goals and statin initiation thresholds based on the risk categories proposed by LAI in 2020.

Risk Group	Treatment Goals		Consider Drug T	herapy
	LDL-C (mg/dl)	Non-HDL (mg/dl)	LDL-C (mg/dl)	Non-HDL (mg/dl)
Extreme Risk Group Category A	<50 (Optional goal	< 80 (Optional goal	>OR = 50	>OR = 80
	$\langle OR = 30 \rangle$	< OR = 60)		
Extreme Risk Group Category B	<OR = 30	< OR = 60	> 30	>60
Very High Risk	<50	<80	>OR= 50	>OR= 80
High Risk	<70	<100	>OR= 70	>OR= 100

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CODE/NAME & ADDRESS: C000138363

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F-703, LADO SARAI, MEHRAULISOUTH WEST

DELHI

NEW DELHI 110030 8800465156 ACCESSION NO: **0031XC020455**PATIENT ID : TANIF22098231

SPIENT BALLENT ID:

AGE/SEX :41 Years Female DRAWN :22/03/2024 10:22:00

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Test Report Status	Final	Results	Biological Reference Interval	Units
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Moderate Risk	<100	<130	>OR= 100	>OR= 130
Low Risk	<100	<130	>OR= 130*	>OR= 160

^{*}After an adequate non-pharmacological intervention for at least 3 months.

References: Management of Dyslipidaemia for the Prevention of Stroke: Clinical Practice Recommendations from the Lipid Association of India. Current Vascular Pharmacology, 2022, 20, 134-155.

LIVER FUNCTION PROFILE, SERUM

BILIRUBIN, TOTAL	0.50	0.2 - 1.2	mg/dL
METHOD: DIAZONIUM SALT			
BILIRUBIN, DIRECT	0.16	0.0 - 0.5	mg/dL
METHOD: DIAZO REACTION			
BILIRUBIN, INDIRECT	0.34	0.1 - 1.0	mg/dL
METHOD : CALCULATED			
TOTAL PROTEIN	8.1	6.0 - 8.30	g/dL
METHOD: BIURET			
ALBUMIN	4.8	3.5 - 5.2	g/dL
METHOD: COLORIMETRIC (BROMCRESOL GREEN)			
GLOBULIN	3.3	2.0 - 3.5	g/dL
ALBUMIN/GLOBULIN RATIO	1.5	1 - 2.1	RATIO
METHOD: CALCULATED PARAMETER			
ASPARTATE AMINOTRANSFERASE(AST/SGOT)	16	5 - 34	U/L
METHOD: ENZYMATIC (NADH (WITHOUT P-5'-P)			
ALANINE AMINOTRANSFERASE (ALT/SGPT)	12	0 - 55	U/L
METHOD: ENZYMATIC (NADH (WITHOUT P-5'-P)			
ALKALINE PHOSPHATASE	78	40 - 150	U/L
METHOD: PARA-NITROPHENYL PHOSPHATE			
GAMMA GLUTAMYL TRANSFERASE (GGT)	21	8 -33	U/L
METHOD: L-GAMMA-GLUTAMYL-4-NITROANALIDE/GLYCYLGLYCINE	KINETIC METHOD		
LACTATE DEHYDROGENASE	146	125 - 220	U/L
METHOD: IFCC LACTATE TO PYRUVATE			

BLOOD UREA NITROGEN (BUN), SERUM

BLOOD UREA NITROGEN 9 7.0 - 18.7 mg/dL

METHOD: UREASE METHOD

CREATININE, SERUM

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Pathologist



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	<u> </u>	<u> </u>	
Test Report Status <u>Final</u>	Results	Biological Reference 1	Interval Units
CREATININE METHOD: KINETIC ALKALINE PICRATE	0.72	0.50 - 1.10	mg/dL
BUN/CREAT RATIO	10.50	50.450	
BUN/CREAT RATIO	12.50	5.0 - 15.0	
URIC ACID, SERUM			4.0
URIC ACID METHOD: URICASE	4.9	2.6 - 6.0	mg/dL
TOTAL PROTEIN, SERUM			
TOTAL PROTEIN METHOD: BIURET	8.1	6.0 - 8.3	g/dL
ALBUMIN, SERUM			
ALBUMIN METHOD: COLORIMETRIC (BROMCRESOL GREEN)	4.8	3.5 - 5.2	g/dL
GLOBULIN			
GLOBULIN METHOD: CALCULATED PARAMETER	3.3	2.0 - 3.5	g/dL
ELECTROLYTES (NA/K/CL), SERUM			
SODIUM, SERUM	138	136 - 145	mmol/L
METHOD: ION SELECTIVE ELECTRODE TECHNOLOGY INDIRECT POTASSIUM, SERUM	4.40	3.5 - 5.1	mmol/L

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METHOD: ION SELECTIVE ELECTRODE TECHNOLOGY INDIRECT

CHLORIDE, SERUM 105 98 - 107 mmol/L

METHOD: ION SELECTIVE ELECTRODE TECHNOLOGY INDIRECT

Interpretation(s)

Sodium	Potassium	Chloride
Decreased in: CCF, cirrhosis, vomiting, diarrhea, excessive sweating, salt-losing nephropathy, adrenal insufficiency, nephrotic syndrome, water intoxication, SIADH. Drugs: thiazides, diuretics, ACE inhibitors, chlorpropamide, carbamazepine, anti depressants (SSRI), antipsychotics.	Decreased in: Low potassium intake, prolonged vomiting or diarrhea, RTA types I and II, hyperaldosteronism, Cushing's syndrome, osmotic diuresis (e.g., hyperglycemia), alkalosis, familial periodic paralysis, trauma (transient). Drugs: Adrenergic agents, diuretics.	Decreased in: Vomiting, diarrhea, renal failure combined with salt deprivation, over-treatment with diuretics, chronic respiratory acidosis, diabetic ketoacidosis, excessive sweating, SIADH, salt-losing nephropathy, porphyria, expansion of extracellular fluid volume, adrenalinsufficiency, hyperaldosteronism, metabolic alkalosis. Drugs: chronic laxative, corticosteroids, diuretics.
Increased in: Dehydration (excessivesweating, severe vomiting or diarrhea),diabetes mellitus, diabetesinsipidus, hyperaldosteronism, inadequate water intake. Drugs: steroids, licorice,oral contraceptives.	Increased in: Massive hemolysis, severe tissue damage, rhabdomyolysis, acidosis, dehydration,renal failure, Addison's disease, RTA type IV, hyperkalemic familial periodic paralysis. Drugs: potassium salts, potassium- sparing diuretics,NSAIDs, beta-blockers, ACE inhibitors, highdose trimethoprim-sulfamethoxazole.	Increased in: Renal failure, nephrotic syndrome, RTA, dehydration, overtreatment with saline, hyperparathyroidism, diabetes insipidus, metabolic acidosis from diarrhea (Loss of HCO3-), respiratory alkalosis, hyperadrenocorticism. Drugs: acetazolamide, androgens, hydrochlorothiazide, salicylates.
Interferences: Severe lipemia or hyperproteinemi, if sodium analysis involves a dilution step can cause spurious results. The serum sodium falls about 1.6 mEq/L for each 100 mg/dL increase in blood glucose.	Interferences: Hemolysis of sample, delayed separation of serum, prolonged fist clenching during blood drawing, and prolonged tourniquet placement. Very high WBC/PLT counts may cause spurious. Plasma potassium levels are normal.	Interferences:Test is helpful in assessing normal and increased anion gap metabolic acidosis and in distinguishing hypercalcemia due to hyperparathyroidism (high serum chloride) from that due to malignancy (Normal serum chloride)

Interpretation(s)

GLUCOSE FASTING, FLUORIDE PLASMA-TEST DESCRIPTION

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.

Decreased in:Pancreatic islet cell disease with increased insulin,insulinoma,adrenocortical insufficiency,hypopituitarism,diffuse liver disease,

pecreased in Participant islet cell disease with increased insulininsulinona, adrenocordical insulincency, hypopicularism, direct 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.

sulfonylureas, tolbutamide, and other oral hypoglycemic agents.

NOTE: While random serum glucose levels correlate with home glucose monitoring results (weekly mean capillary glucose values), there is wide fluctuation within individuals. Thus, glycosylated hemoglobin(HbA1c) levels are favored to monitor glycemic control.

individuals. Thus, 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 Alignetary, Hypoglycaemia Increased in the control of the contr

index & response to food consumed, Alimentary Hypoglycemia, Increased insulin response & sensitivity etc.

GLUCOSE, POST-PRANDIAL, PLASMA-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. Additional test HbA1c

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West Bengal, India







REF. DOCTOR: DR. ARCOFEMI HEALTHCARE LTD **PATIENT NAME: TANIMA LAMA** (MEDIWHEEL

CODE/NAME & ADDRESS : C000138363 ARCOFEMI HEALTHCARE LTD (MEDIWHEEL F-703, LADO SARAI, MEHRAULISOUTH WEST

DELHI

NEW DELHI 110030 8800465156

ACCESSION NO: 0031XC020455 PATIENT ID : TANIF22098231

CHIENT BATTENT ID:

:41 Years AGE/SEX Female DRAWN :22/03/2024 10:22:00

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

LIVER FUNCTION PROFILE, SERUM-

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, Pagets disease, Rickets, Sarcoidosis etc. Lower-than-normal ALP levels seen

in Hypophosphatasia, Malnutrition, Protein deficiency, Wilsons 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.

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, Waldenstroms 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** 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), 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, Muscuophy
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-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, Waldenstroms 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.

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Test Report Status Final Results Biological Reference Interval Units

SPECIALISED CHEMISTRY - HORMONE

MEDI WHEEL FULL BODY HEALTH CHECKUP ABOVE 40FEMALE

THYROID PANEL, SERUM

T3 77.7 Non-Pregnant Women 35 - 199/dL

Pregnant Women

1st Trimester:105.0 - 230.0 2nd Trimester:129.0 - 262.0 3rd Trimester:135.0 - 262.0

METHOD: TWO-STEP CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY

T4 6.66 Non-Pregnant Women µg/dL

4.87 - 11.71 Pregnant Women

1st Trimester: 7.33 - 14.80 2nd Trimester: 7.93 - 16.10 3rd Trimester: 6.95 - 15.70

METHOD: TWO-STEP CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY

TSH (ULTRASENSITIVE) 3.074 Non-Pregnant Women 0.35 - µIU/mL

4.94

Pregnant Women (As per American Thyroid Association) 1st Trimester 0.100 - 2.500 2nd Trimester 0.200 - 3.000 3rd Trimester 0.300 - 3.000

METHOD: TWO-STEP CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY

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, TSH levels are significantly elevated, while in secondary and tertiary hyperthyroidism, TSH levels are low. Below mentioned are the guidelines for Pregnancy related reference ranges for Total T4, TSH & Total T3.Measurement of the serum TT3 level is a more sensitive test for the diagnosis of hyperthyroidism, 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

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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, Iodine 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 Hyperthyroidism
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.

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

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Test Report Status Final Results Biological Reference Interval Units

CONDITIONS OF LABORATORY TESTING & REPORTING

- 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 AGILUS Directory of Services.
- 3. Result delays could occur due to unforeseen circumstances such as non-availability of kits / equipment breakdown / natural calamities / technical downtime or any other unforeseen event.
- 4. A requested test might not be performed if:
 - i. Specimen received is insufficient or inappropriate
 - ii. Specimen quality is unsatisfactory
 - iii. Incorrect specimen type
 - iv. Discrepancy between identification on specimen container label and test requisition form

- 5. AGILUS Diagnostics confirms that all tests have been performed or assayed with highest quality standards, clinical safety & technical integrity.
- 6. Laboratory results should not be interpreted in isolation; it must be correlated with clinical information and be interpreted by registered medical practitioners only to determine final diagnosis.
- 7. Test results may vary based on time of collection, physiological condition of the patient, current medication or nutritional and dietary changes. Please consult your doctor or call us for any clarification.
- 8. Test results cannot be used for Medico legal purposes.
- 9. In case of queries please call customer care (91115 91115) within 48 hours of the report.

Agilus Diagnostics Ltd

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