

Male

PATIENT NAME: MR. RAKESH BHAGESHWAR NANDANWAR REF. DOCTOR: SELF

CODE/NAME & ADDRESS : C000138394

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

DELHÍ

NEW DELHI 110030 8800465156 ACCESSION NO : **0181WJ001010** 

PATIENT ID : FH.8178815

CLIENT PATIENT ID: ABHA NO : DRAWN :31/10/2023 09:00:00
RECEIVED :31/10/2023 09:07:40

:33 Years

AGE/SEX

REPORTED :01/11/2023 13:17:10

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

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

**XRAY-CHEST** 

IMPRESSION NO ABNORMALITY DETECTED

**ECG** 

ECG WITHIN NORMAL LIMITS

**MEDICAL HISTORY** 

RELEVANT PRESENT HISTORY C/O CHEST INFECTION 5 DAYS.

RELEVANT PAST HISTORY COVID TWICE IN THE PAST. HOME QUARANTINED.

RELEVANT PERSONAL HISTORY MARRIED / MIXED DIET / NO ALLERGIES / NO SMOKING / ALCOHOL.

RELEVANT FAMILY HISTORY NOT SIGNIFICANT HISTORY OF MEDICATIONS NOT SIGNIFICANT

**ANTHROPOMETRIC DATA & BMI** 

HEIGHT IN METERS 1.70 mts
WEIGHT IN KGS. 74 Kgs

BMI 26 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

#### **GENERAL EXAMINATION**

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

STATUS

BUILT / SKELETAL FRAMEWORK
FACIAL APPEARANCE
SKIN
UPPER LIMB
LOWER LIMB
NORMAL
NECK
NORMAL

NECK LYMPHATICS / SALIVARY GLANDS NOT ENLARGED OR TENDER

THYROID GLAND NOT ENLARGED

CAROTID PULSATION NORMAL TEMPERATURE NORMAL

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PULSE 76/MINREGULAR, ALL PERIPHERAL PULSES WELL FELT, NO CAROTID

BRUIT

RESPIRATORY RATE NORMAL

CARDIOVASCULAR SYSTEM

BP 122/80 MM HG mm/Hg

(SUPINE) NORMAL

**ABSENT** 

PERICARDIUM NORMAL
APEX BEAT NORMAL
HEART SOUNDS NORMAL
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** 

**HERNIA** 

APPEARANCE NORMAL VENOUS PROMINENCE ABSENT

LIVER NOT PALPABLE SPLEEN NOT PALPABLE

**CENTRAL NERVOUS SYSTEM** 

HIGHER FUNCTIONS

CRANIAL NERVES

CEREBELLAR FUNCTIONS

SENSORY SYSTEM

MOTOR SYSTEM

REFLEXES

NORMAL

NORMAL

NORMAL

**MUSCULOSKELETAL SYSTEM** 

SPINE NORMAL JOINTS NORMAL

**BASIC EYE EXAMINATION** 

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**NORMAL** CONJUNCTIVA **NORMAL EYELIDS** EYE MOVEMENTS **NORMAL CORNEA NORMAL** 

DISTANT VISION RIGHT EYE WITHOUT

GLASSES

DISTANT VISION LEFT EYE WITHOUT

**GLASSES** 

NEAR VISION RIGHT EYE WITHOUT GLASSES WITHIN NORMAL LIMIT WITHIN NORMAL LIMIT NEAR VISION LEFT EYE WITHOUT GLASSES

COLOUR VISION

**SUMMARY** 

RELEVANT HISTORY RELEVANT GP EXAMINATION FINDINGS REMARKS / RECOMMENDATIONS

NOT SIGNIFICANT NOT SIGNIFICANT TO DO S.IRON STUDIES.

WITHIN NORMAL LIMIT

WITHIN NORMAL LIMIT

REPEAT CBC,TSH,SR.POTASSIUM AFTER 3 MONTHS OF DIET AND

EXERCISE.

**NORMAL** 

AVOID HIGH POTASSIUM CANTAINING FOODS.

LIKE BANANAS, COCOUNT WATER.

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Agilus Diagnostics Ltd. S.K. Tower, Hari Niwas, Lbs Marg Thane, 400602 Maharashtra, India

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**ULTRASOUND ABDOMEN** 

**ULTRASOUND ABDOMEN** 

NO ABNORMALITIES DETECTED

TMT OR ECHO

CLINICAL PROFILE

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.

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

#### **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.
- 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**

Fortis Hospital, Sector 62, Phase VIII, Mohali 160062

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HAEMATOLOGY - CBC				
MEDI WHEEL FULL BODY HEALTH CHECK UP BI	LOW 40 MALE			
BLOOD COUNTS, EDTA WHOLE BLOOD				
HEMOGLOBIN (HB) METHOD: SLS- HEMOGLOBIN DETECTION METHOD	12.5 Low	13.0 - 17.0	g/dL	
RED BLOOD CELL (RBC) COUNT METHOD: HYDRODYNAMIC FOCUSING BY DC DETECTION	6.12 High	4.5 - 5.5	mil/μL	
WHITE BLOOD CELL (WBC) COUNT METHOD: FLUORESCENCE FLOW CYTOMETRY	6.67	4.0 - 10.0	thou/µL	
PLATELET COUNT  METHOD: HYDRODYNAMIC FOCUSING BY DC DETECTION	191	150 - 410	thou/µL	
RBC AND PLATELET INDICES				
HEMATOCRIT (PCV)  METHOD: CUMULATIVE PULSE HEIGHT DETECTION METHOD	40.8	40.0 - 50.0	%	
MEAN CORPUSCULAR VOLUME (MCV) METHOD: CALCULATED FROM RBC & HCT	66.7 Low	83.0 - 101.0	fL	
MEAN CORPUSCULAR HEMOGLOBIN (MCH)  METHOD: CALCULATED FROM THE RBC & HGB	20.4 Low	27.0 - 32.0	pg	
MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION (MCHC) METHOD: CALCULATED FROM THE HGB & HCT	30.6 Low	31.5 - 34.5	g/dL	
RED CELL DISTRIBUTION WIDTH (RDW)  METHOD: CALCULATED FROM RBC SIZE DISTRIBUTION CURVE	18.3 High	11.6 - 14.0	%	
MENTZER INDEX	10.9			
WBC DIFFERENTIAL COUNT				
NEUTROPHILS	58	40 - 80	%	
METHOD: FLOW CYTOMETRY WITH LIGHT SCATTERING				
LYMPHOCYTES	33	20 - 40	%	
METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING				
MONOCYTES  METHOD: FLOW CYTOMETRY WITH LIGHT SCATTERING	7	2 - 10	%	
EOSINOPHILS	2	1 - 6	%	
METHOD: FLOW CYTOMETRY WITH LIGHT SCATTERING	-	1 0		
BASOPHILS  METHOD: FLOW CYTOMETRY WITH LIGHT SCATTERING	0	0 - 1	%	

BWindshift.

Dr.Priyal Chinchkhede, MD Consultant Pathologist





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Test Report Status <u>Final</u>	Results	Biological Reference	Interval Units
ABSOLUTE NEUTROPHIL COUNT	3.87	2.0 - 7.0	thou/μL
METHOD: FLOW CYTOMETRY WITH LIGHT SCATTERING			
ABSOLUTE LYMPHOCYTE COUNT	2.22	1.0 - 3.0	thou/µL
METHOD: FLOW CYTOMETRY WITH LIGHT SCATTERING			
ABSOLUTE MONOCYTE COUNT	0.45	0.2 - 1.0	thou/µL
METHOD: FLOW CYTOMETRY WITH LIGHT SCATTERING			
ABSOLUTE EOSINOPHIL COUNT	0.13	0.02 - 0.50	thou/µL
METHOD: FLOW CYTOMETRY WITH LIGHT SCATTERING			
ABSOLUTE BASOPHIL COUNT	0 Low	0.02 - 0.10	thou/µL
METHOD: FLOW CYTOMETRY WITH LIGHT SCATTERING			
NEUTROPHIL LYMPHOCYTE RATIO (NLR)	1.7		
MORPHOLOGY			
RBC	MICROCYTOSIS A	ND ANISOCYTOSIS	
WBC	NORMAL MORPHO	LOGY	
METHOD: MICROSCOPIC EXAMINATION			
PLATELETS	ADEQUATE		

BLOOD COUNTS, EDTA WHOLE BLOOD-The cell morphology is well preserved for 24hrs. However after 24-48 hrs a progressive increase in MCV and HCT is observed leading to a decrease in MCHC. A direct smear is recommended for an accurate differential count and for examination of RBC morphology.

RBC AND PLATELET INDICES-Mentzer index (MCV/RBC) is an automated cell-counter based calculated screen tool to differentiate cases of Iron deficiency anaemia(>13) from Beta thalassaemia trait

(<13) in patients with microcytic anaemia. This needs to be interpreted in line with clinical correlation and suspicion. Estimation of HbA2 remains the gold standard for diagnosing a case of beta thalassaemia trait.

WBC DIFFERENTIAL COUNT-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.

ENTARRAGE

Dr. Priyal Chinchkhede, MD **Consultant Pathologist** 





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#### **HAEMATOLOGY**

Results

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

# ERYTHROCYTE SEDIMENTATION RATE (ESR), WHOLE BLOOD

E.S.R 3 0 - 14 mm

METHOD: MODIFIED WESTERGREN

GLYCOSYLATED HEMOGLOBIN(HBA1C), EDTA WHOLE BLOOD

HBA1C 4.7 Non-diabetic Adult < 5.7 %

Pre-diabetes 5.7 - 6.4

(ADA Guideline 2021)

Diabetes diagnosis: > or = 6.5 Therapeutic goals: < 7.0 Action suggested : > 8.0

METHOD: HPLC

ESTIMATED AVERAGE GLUCOSE(EAG) 88.2 < 116.0 mg/dL

METHOD: CALCULATED PARAMETER



Interpretation(s)

BWindshill.

Dr.Priyal Chinchkhede, MD Consultant Pathologist



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ERYTHROCYTE SEDIMENTATION RATE (ESR), WHOLE 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)

#### REFERENCE :

- 1. Nathan and Oski's Haematology of Infancy and Childhood, 5th edition 2. Paediatric reference intervals. AACC Press, 7th edition. Edited by S. Soldin 3. The reference for the adult reference range is "Practical Haematology by Dacie and Lewis,10th edition. GLYCOSYLATED HEMOGLOBIN(HBA1C), EDTA WHOLE BLOOD-**Used For**:
- 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.

- eAG (Estimated average glucose) converts percentage HbA1c to md/dl, to compare blood glucose levels.
   eAG gives an evaluation of blood glucose levels for the last couple of months.
   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

Philadelphia

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#### **IMMUNOHAEMATOLOGY**

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

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

**ABO GROUP** TYPE O

METHOD: GEL COLUMN AGGLUTINATION METHOD.

RH TYPE **POSITIVE** 

METHOD: GEL COLUMN AGGLUTINATION 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.

EWindshield

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#### **BIOCHEMISTRY**

# MEDI WHEEL FULL BODY HEALTH CHECK UP BELOW 40 MALE

GLUCOSE FASTING, FLUORIDE PLASMA

FBS (FASTING BLOOD SUGAR)

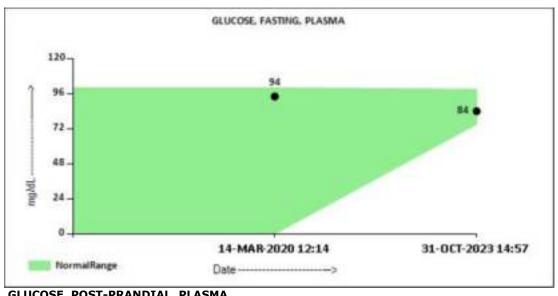
84

Normal 75 - 99

mg/dL

Pre-diabetics: 100 - 125 Diabetic: > or = 126

METHOD: ENZYMATIC REFERENCE METHOD WITH HEXOKINASE



**GLUCOSE, POST-PRANDIAL, PLASMA** 

PPBS(POST PRANDIAL BLOOD SUGAR)

METHOD: ENZYMATIC REFERENCE METHOD WITH HEXOKINASE

79

70 - 139

mg/dL

Dr. Ushma Wartikar, MD **Consultant Pathologist** 

Bhlachthair

Dr. Priyal Chinchkhede, MD **Consultant Pathologist** 

Dr.(Mrs)Neelu K Bhojani Lab Head





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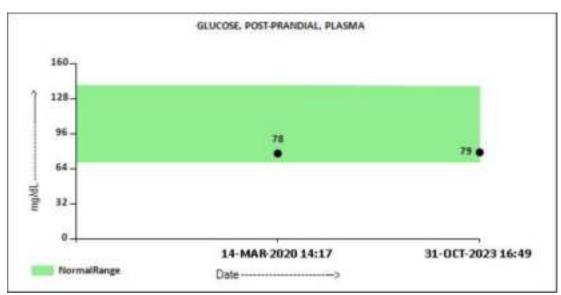
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LIPID PROFILE WITH CALCULATED LDL

CHOLESTEROL, TOTAL 109 Desirable: < 200 mg/dL

Borderline : 200 - 239 High : > / = 240

METHOD: ENZYMATIC COLORIMETRIC ASSAY

TRIGLYCERIDES 72 Normal: < 150 mg/dL

Borderline high: 150 - 199

High: 200 - 499 Very High: >/= 500

METHOD: ENZYMATIC COLORIMETRIC ASSAY

METHOD: ENZYMATIC, COLORIMETRIC

HDL CHOLESTEROL **30 Low** At Risk: < 40 mg/dL

Desirable: > or = 60

CHOLESTEROL LDL 65 Adult levels:

Adult levels: mg/dL

Optimal < 100

Near optimal/above optimal:

100-129

Borderline high: 130-159

High: 160-189Very high: = 190

METHOD: ENZYMATIC COLORIMETRIC ASSAY

Dr. Ushma Wartikar, MD Consultant Pathologist ENTARKHILL.

Dr.Priyal Chinchkhede, MD Consultant Pathologist Alsimi

Dr.(Mrs)Neelu K Bhojani Lab Head



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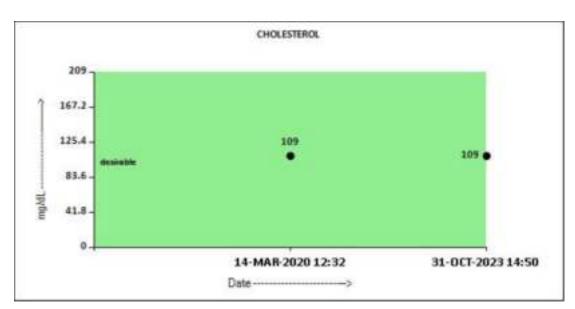
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NON HDL CHOLESTEROL	79	Desirable: < 130 mg/dL Above Desirable: 130 -159 Borderline High: 160 - 189 High: 190 - 219 Very high: > / = 220
VERY LOW DENSITY LIPOPROTEIN	14.4	< OR = 30.0 mg/dL
CHOL/HDL RATIO	3.6	Low Risk: 3.3 - 4.4 Average Risk: 4.5 - 7.0 Moderate Risk: 7.1 - 11.0 High Risk: > 11.0
LDL/HDL RATIO	2.2	0.5 - 3.0 Desirable/Low Risk 3.1 - 6.0 Borderline/Moderate Risk >6.0 High Risk



Dr. Ushma Wartikar, MD Consultant Pathologist EWindschill.

Dr.Priyal Chinchkhede, MD Consultant Pathologist Dr. (Mrs.)Neelu K. B.

Dr.(Mrs)Neelu K Bhojani Lab Head





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Agilus Diagnostics Ltd. Mulund Goregoan Link Road Mumbai, 400078 Maharashtra, India Fax:







CODE/NAME & ADDRESS : C000138394
ARCOFEMI HEALTHCARE LTD (MEDIWHEEL

F-703, F-703, LADO SARAI, MEHRAULISOUTH WEST

<u>Final</u>

DELHI

NEW DELHI 110030 8800465156

**Test Report Status** 

ACCESSION NO: 0181WJ001010

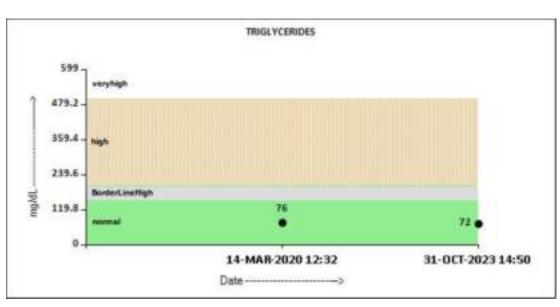
PATIENT ID : FH.8178815

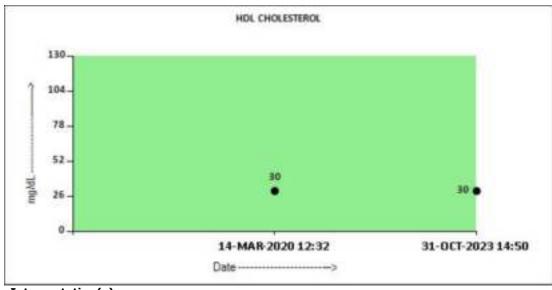
CLIENT PATIENT ID: ABHA NO :

Results

AGE/SEX :33 Years Male
DRAWN :31/10/2023 09:00:00
RECEIVED :31/10/2023 09:07:40
REPORTED :01/11/2023 13:17:10

Biological Reference Interval Units





Interpretation(s)

Dr. Ushma Wartikar, MD Consultant Pathologist Entrachenia

Dr.Priyal Chinchkhede, MD Consultant Pathologist Dr. (Mrs.) Nagly K

Dr.(Mrs)Neelu K Bhojani Lab Head Page 13 Of 25





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Agilus Diagnostics Ltd. Mulund Goregoan Link Road Mumbai, 400078 Maharashtra, India







Male

PATIENT NAME: MR. RAKESH BHAGESHWAR NANDANWAR **REF. DOCTOR: SELF** 

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DELHI

**NEW DELHI 110030** 8800465156

ACCESSION NO: 0181WJ001010

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:33 Years

AGE/SEX

REPORTED :01/11/2023 13:17:10

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

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

	reservo (reneroscerotte em diovinscumi di	the contract of the contract o	
Risk Category			
Extreme risk group	A.CAD with > 1 feature of high risk group		
	B. CAD with > 1 feature of Very high risk g	roup 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	1	
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			
Age > or = 45 years in males and > or = 55 years in females     Current Cigarette smoking or tobacco use			
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	ent Goals		Consider Drug Therapy	
	LDL-C (mg/dl)	Non-HDL (mg/dl)	LDL-C (mg/dl)	Non-HDL (mg/df)	
Extreme Risk Group Category A	<50 (Optional goal < OR = 30 )	< 80 (Optional goal <or 60)<="" =="" td=""><td>&gt;OR = 50</td><td>&gt;OR = 80</td></or>	>OR = 50	>OR = 80	
Extreme Risk Group Category B	<or 30<="" =="" td=""><td><or 60<="" =="" td=""><td>&gt; 30</td><td>&gt;60</td></or></td></or>	<or 60<="" =="" td=""><td>&gt; 30</td><td>&gt;60</td></or>	> 30	>60	
Very High Risk	<50	<80	>OR= 50	>OR= 80	
High Risk	<70	<100	>OR= 70	>OR= 100	
Moderate Risk	<100	<130	>OR= 100	>OR= 130	
Low Risk	<100	<130	>OR= 130*	>OR= 160	

<sup>\*</sup>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  METHOD: COLORIMETRIC DIAZO	1.30 High	Upto 1.2	mg/dL
BILIRUBIN, DIRECT	0.93 High	< 0.30	mg/dL
BILIRUBIN, INDIRECT	0.37	0.1 - 1.0	mg/dL
TOTAL PROTEIN  METHOD: COLORIMETRIC	8.7 High	6.0 - 8.0	g/dL
ALBUMIN METHOD: COLORIMETRIC	5.5 High	3.97 - 4.94	g/dL
GLOBULIN	3.2	2.0 - 3.5	g/dL

Dr. Ushma Wartikar, MD **Consultant Pathologist** 

EMINDEREN

Dr. Priyal Chinchkhede, MD **Consultant Pathologist** 

Dr.(Mrs)Neelu K Bhojani Lab Head





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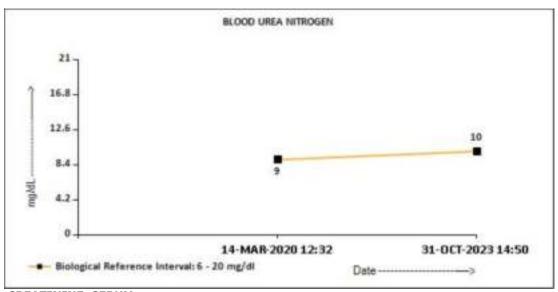
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Test Report Status <u>Final</u>	Results	Biological Reference Interva	al Units
ALBUMIN/GLOBULIN RATIO	1.7	1.0 - 2.1	RATIO
ASPARTATE AMINOTRANSFERASE(AST/SGOT) METHOD: UV ABSORBANCE	25	< OR = 50	U/L
ALANINE AMINOTRANSFERASE (ALT/SGPT)  METHOD: UV ABSORBANCE	34	< OR = 50	U/L
ALKALINE PHOSPHATASE  METHOD: COLORIMETRIC	76	40 - 129	U/L
GAMMA GLUTAMYL TRANSFERASE (GGT)  METHOD: ENZYMATIC, COLORIMETRIC	18	0 - 60	U/L
LACTATE DEHYDROGENASE  METHOD: UV ABSORBANCE	171	125 - 220	U/L
BLOOD UREA NITROGEN (BUN), SERUM			
BLOOD UREA NITROGEN  METHOD: ENZYMATIC ASSAY	10	6 - 20	mg/dL



**CREATININE, SERUM** 

CREATININE 0.84 0.7 - 1.2 mg/dL

METHOD: COLORIMETRIC

Dr. Ushma Wartikar, MD

**Consultant Pathologist** 

Philadelphia.

Dr.Priyal Chinchkhede, MD Consultant Pathologist Dr.(Mrs)Neelu K Bhojani Lab Head Page 15 Of 25





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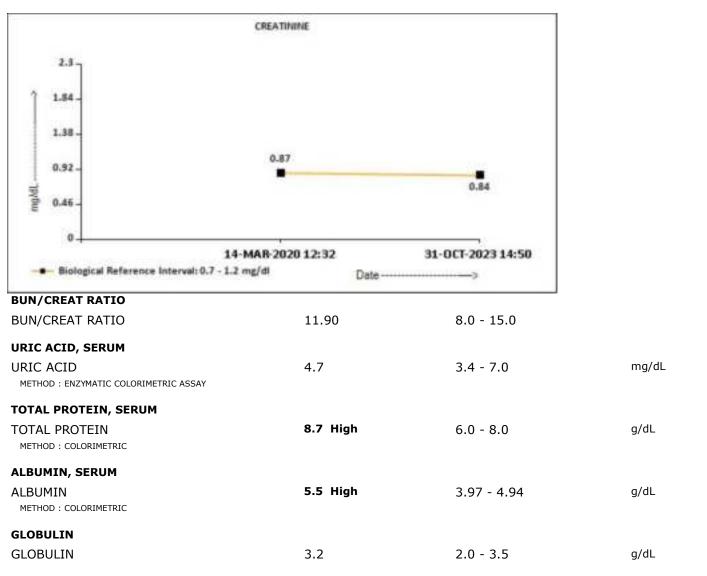
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Test Report Status <u>Final</u> Results Biological Reference Interval Units



**ELECTROLYTES (NA/K/CL), SERUM** 

Dr. Ushma Wartikar, MD Consultant Pathologist ENTARKABL.

Dr.Priyal Chinchkhede, MD Consultant Pathologist Mary Markey

Dr.(Mrs)Neelu K Bhojani Lab Head

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:33 Years

AGE/SEX

REPORTED :01/11/2023 13:17:10

Test Report Status <u>Final</u>	Results	Biological Reference	e Interval Units
SODIUM, SERUM	139	136 - 145	mmol/L
METHOD: ION SELECTIVE ELECTRODE TECHNOLOGY			
POTASSIUM, SERUM	5.55 High	3.5 - 5.1	mmol/L
METHOD: ION SELECTIVE ELECTRODE TECHNOLOGY			
CHLORIDE, SERUM	103	98 - 107	mmol/L
METHOD: ION SELECTIVE ELECTRODE TECHNOLOGY			

#### Interpretation(s)

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

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, 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: 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.

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.

Dr. Ushma Wartikar, MD **Consultant Pathologist** 

Christenski

Dr. Priyal Chinchkhede, MD **Consultant Pathologist** 

Dr.(Mrs)Neelu K Bhojani Lab Head





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DELHI

**NEW DELHI 110030** 

8800465156

ACCESSION NO : 0181WJ001010

PATIENT ID : FH.8178815

CLIENT PATIENT ID: ABHA NO

:31/10/2023 09:00:00 DRAWN RECEIVED: 31/10/2023 09:07:40

:33 Years

AGE/SEX

REPORTED :01/11/2023 13:17:10

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

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 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.

Dr. Ushma Wartikar, MD **Consultant Pathologist** 

Bhindehna

Dr. Prival Chinchkhede, MD Consultant Pathologist

Dr.(Mrs)Neelu K Bhojani Lab Head



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

#### **CLINICAL PATH - URINALYSIS**

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

PHYSICAL EXAMINATION, URINE

COLOR PALE YELLOW

METHOD: MICROSCOPIC EXAMINATION

APPEARANCE CLEAR

METHOD: MICROSCOPIC EXAMINATION

CHEMICAL EXAMINATION, URINE

PH 6.0 5.00 - 7.50

METHOD: METHYL RED & BROMOTHYMOL BLUE

SPECIFIC GRAVITY 1.005 Low 1.010 - 1.030 PROTEIN NOT DETECTED NOT DETECTED

METHOD: TETRA BROMOPHENOL BLUE/SULFOSALICYLIC ACID

GLUCOSE NOT DETECTED NOT DETECTED

METHOD: GLUCOSE OXIDASE / PEROXIDASE (GOD - POD) METHOD

KETONES NOT DETECTED NOT DETECTED

METHOD: SODIUM NITROPRUSSIDE REACTION

BLOOD NOT DETECTED NOT DETECTED

METHOD: STRIP TEST - DIAZONIUM SALT COUPLING

UROBILINOGEN NORMAL NORMAL

METHOD : CAFFEINE BENZOATE

NITRITE NOT DETECTED NOT DETECTED

METHOD: STRIP NAPHTHOETHYLENEDIAMINE HYDROCHOLORIDE, TATTANIC ACID

LEUKOCYTE ESTERASE NOT DETECTED NOT DETECTED

METHOD: STRIP HETROCYCLIC CARBOXYLIC ACID ESTER, DIAZONIUM SALT

MICROSCOPIC EXAMINATION, URINE

RED BLOOD CELLS NOT DETECTED NOT DETECTED /HPF

METHOD: MICROSCOPIC EXAMINATION

PUS CELL (WBC'S) 0-1 0-5 /HPF

METHOD: MICROSCOPIC EXAMINATION

EPITHELIAL CELLS 1-2 0-5 /HPF

METHOD: MICROSCOPIC EXAMINATION

CASTS NOT DETECTED

METHOD: MICROSCOPIC EXAMINATION

CRYSTALS NOT DETECTED

ENTERNANCE

Dr.Priyal Chinchkhede, MD Consultant Pathologist Dr. Ushma Wartikar, MD Consultant Pathologist

Dr. (Mrc.)Noolu K Bl

Dr.(Mrs)Neelu K Bhojani Lab Head



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METHOD: MICROSCOPIC EXAMINATION

BACTERIA NOT DETECTED NOT DETECTED

METHOD: MICROSCOPIC EXAMINATION

YEAST NOT DETECTED NOT DETECTED

#### Interpretation(s)

The following table describes the probable conditions, in which the analytes are present in urine

Presence of	Conditions			
Proteins	Inflammation or immune illnesses			
Pus (White Blood Cells)	Urinary tract infection, urinary tract or kidney stone, tumors or any kind			
	of kidney impairment			
Glucose	Diabetes or kidney disease			
Ketones	Diabetic ketoacidosis (DKA), starvation or thirst			
Urobilinogen	Liver disease such as hepatitis or cirrhosis			
Blood	Renal or genital disorders/trauma			
Bilirubin	Liver disease			
Erythrocytes	Urological diseases (e.g. kidney and bladder cancer, urolithiasis), urinary			
	tract infection and glomerular diseases			
Leukocytes	Urinary tract infection, glomerulonephritis, interstitial nephritis either			
	acute or chronic, polycystic kidney disease, urolithiasis, contamination by			
	genital secretions			
Epithelial cells	Urolithiasis, bladder carcinoma or hydronephrosis, ureteric stents or			
	bladder catheters for prolonged periods of time			
Granular Casts	Low intratubular pH, high urine osmolality and sodium concentration,			
	interaction with Bence-Jones protein			
Hyaline casts	Physical stress, fever, dehydration, acute congestive heart failure, renal			
	diseases			
Calcium oxalate	Metabolic stone disease, primary or secondary hyperoxaluria, intravenous			
	infusion of large doses of vitamin C, the use of vasodilator naftidrofuryl			
	oxalate or the gastrointestinal lipase inhibitor orlistat, ingestion of			
	ethylene glycol or of star fruit (Averrhoa carambola) or its juice			
Uric acid	arthritis			
Bacteria	Urinary infectionwhen present in significant numbers & with pus cells.			
Trichomonas vaginalis	Vaginitis, cervicitis or salpingitis			

ENTARKARE.

Dr.Priyal Chinchkhede, MD Consultant Pathologist Dr. Ushma Wartikar, MD Consultant Pathologist

Dr.(Mrs)Neelu K Bhojani Lab Head

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

**CLINICAL PATH - STOOL ANALYSIS** 

MEDI WHEEL FULL BODY HEALTH CHECK UP BELOW 40 MALE

PHYSICAL EXAMINATION, STOOL

COLOUR BROWN

 ${\tt METHOD}: {\tt VISUAL}$ 

CONSISTENCY SEMI FORMED

METHOD: VISUAL

MUCUS NOT DETECTED NOT DETECTED

METHOD: VISUAL

VISIBLE BLOOD ABSENT ABSENT ABSENT

METHOD : VISUAL

CHEMICAL EXAMINATION, STOOL

STOOL PH 6.5

METHOD: USING PH PAPER

OCCULT BLOOD NOT DETECTED NOT DETECTED

METHOD: HEMOSPOT

MICROSCOPIC EXAMINATION, STOOL

PUS CELLS 1-2 /hpf

RED BLOOD CELLS NOT DETECTED NOT DETECTED /HPF

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

TROPHOZOITES NOT DETECTED NOT DETECTED

METHOD: MICROSCOPIC EXAMINATION

FAT ABSENT VEGETABLE CELLS ABSENT

CONCENTRATION METHOD NO OVA & CYST SEEN AFTER PERFORMING CONCENTRATION

TECHNIQUE FOR STOOL SAMPLE.

Interpretation(s)

Stool routine analysis is only a screening test for disorders of gastrointentestinal tract like infection, malabsorption, etc.The following

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<u>Final</u>

DELHI

NEW DELHI 110030

**Test Report Status** 

8800465156

ACCESSION NO : **0181WJ001010** 

PATIENT ID : FH.8178815

CLIENT PATIENT ID: ABHA NO :

Results

AGE/SEX :33 Years Male
DRAWN :31/10/2023 09:00:00
RECEIVED :31/10/2023 09:07:40

RECEIVED : 31/10/2023 09:07:40 REPORTED :01/11/2023 13:17:10

Biological Reference Interval Units

#### table describes the probable conditions, in which the analytes are present in stool.

PRESENCE OF	CONDITION	
Pus cells	Pus in the stool is an indication of infection	
Red Blood cells	Parasitic or bacterial infection or an inflammatory bowel condition such as ulcerative colitis	
Parasites	Infection of the digestive system. Stool examination for ova and parasite detects presence of parasitic infestation of gastrointestinal tract. Various forms of parasite that can be detected include cyst, trophozoite and larvae. One negative result does not rule out the possibility of parasitic infestation. Intermittent shedding of parasites warrants examinations of multiple specimens tested on consecutive days. Stool specimens for parasitic examination should be collected before initiation of antidiarrheal therapy or antiparasitic therapy. This test does not detect presence of opportunistic parasites like Cyclospora, Cryptosporidia and Isospora species. Examination of Ova and Parasite has been carried out by direct and concentration techniques.	
Mucus	Mucus is a protective layer that lubricates, protects& reduces damage due to bacteria or viruses.	
Charcot-Leyden crystal	Parasitic diseases.	
Ova & cyst	Ova & cyst indicate parasitic infestation of intestine.	
Frank blood	Bleeding in the rectum or colon.	
Occult blood	Occult blood indicates upper GI bleeding.	
Macrophages	Macrophages in stool are an indication of infection as they are protective cells.	
Epithelial cells	Epithelial cells that normally line the body surface and internal organs show up in stool when there is inflammation or infection.	
Fat	Increased fat in stool maybe seen in conditions like diarrhoea or malabsorption.	
pH	Normal stool pH is slightly acidic to neutral. Breast-fed babies generally have an acidic stool.	

#### ADDITIONAL STOOL TESTS:

- Stool Culture: This test is done to find cause of GI infection, make decision about best treatment for GI infection & to find out if treatment for GI infection worked.
- Fecal Calprotectin: It is a marker of intestinal inflammation. This test is done to differentiate Inflammatory Bowel Disease (IBD) from Irritable Bowel Syndrome (IBS).
- 3. Fecal Occult Blood Test(FOBT): This test is done to screen for colon cancer & to evaluate possible cause of unexplained anaemia.
- Clostridium Difficile Toxin Assay: This test is strongly recommended in healthcare associated bloody or waterydiarrhoea, due to
  overuse of broad spectrum antibiotics which alter the normal GI flora.
- Biofire (Film Array) GI PANEL: In patients of Diarrhoea, Dysentry, Rice watery Stool, FDA approved, Biofire Film Array
  Test, (Real Time Multiplex PCR) is strongly recommended as it identifies organisms, bacteria, fungi, virus, parasite and other
  opportunistic pathogens, Vibrio cholera infections only in 3 hours. Sensitivity 96% & Specificity 99%.
- Rota Virus Immunoassay: This test is recommended in severe gastroenteritis in infants & children associated with watery diarrhoea, vomitting& abdominal cramps. Adults are also affected. It is highly contagious in nature.

Dr. Sheetal Sawant, MD
Consultant Microbiologist

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Dr. Sheetal Sawant, MD Consultant Microbiologist



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Male

PATIENT NAME: MR. RAKESH BHAGESHWAR NANDANWAR REF. DOCTOR: SELF

CODE/NAME & ADDRESS : C000138394

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#### **SPECIALISED CHEMISTRY - HORMONE**

# MEDI WHEEL FULL BODY HEALTH CHECK UP BELOW 40 MALE

#### THYROID PANEL, SERUM

T3 135.0 80 - 200 ng/dL

METHOD: ELECTROCHEMILUMINESCENCE

T4 8.25 5.1 - 14.1  $\mu g/dL$ 

METHOD: ELECTROCHEMILUMINESCENCE

TSH (ULTRASENSITIVE) 5.070 High 0.27 - 4.2 µIU/mL

METHOD: ELECTROCHEMILUMINESCENCE

#### 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 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

Dr. Ushma Wartikar, MD Consultant Pathologist BWindshift.

Dr.Priyal Chinchkhede, MD Consultant Pathologist Misjone

Dr.(Mrs)Neelu K Bhojani Lab Head



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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.

Dr. Ushma Wartikar, MD Consultant Pathologist Philadelphia.

Dr.Priyal Chinchkhede, MD Consultant Pathologist Dr.(Mrs)Neelu K I

Dr.(Mrs)Neelu K Bhojani Lab Head





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