



PATIENT NAME: RANDEEP CHAWLA REF. DOCTOR: SELF

CODE/NAME & ADDRESS: C000138383

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

DELHÍ

NEW DELHI 110030 8800465156 ACCESSION NO : 0080XA008665

PATIENT ID : RANDM05128180

CLIENT PATIENT ID:

AGE/SEX

RECEIVED : 27/01/2024 09:07:27 REPORTED : 27/01/2024 14:27:30

:42 Years

Test Report Status Final Results Biological Reference Interval Units

н	AEMATOLOGY - CBC		
MEDI WHEEL FULL BODY HEALTH CHECK UP A	BOVE 40 MALE		
BLOOD COUNTS,EDTA WHOLE BLOOD			
HEMOGLOBIN (HB)	14.8	13.0 - 17.0	g/dL
METHOD: CYANMETHEMOGLOBIN METHOD			
RED BLOOD CELL (RBC) COUNT METHOD: ELECTRICAL IMPEDANCE	5.46	4.5 - 5.5	mil/µL
WHITE BLOOD CELL (WBC) COUNT	4.72	4.0 - 10.0	thou/µL
METHOD : ELECTRICAL IMPEDANCE			
PLATELET COUNT	325	150 - 410	thou/µL
METHOD : ELECTRICAL IMPEDANCE			
RBC AND PLATELET INDICES			
HEMATOCRIT (PCV)	44.6	40 - 50	%
METHOD : ELECTRICAL IMPEDANCE	81.8 Low	83 - 101	fL
MEAN CORPUSCULAR VOLUME (MCV) METHOD: CALCULATED PARAMETER	81.8 LOW	83 - 101	IL
MEAN CORPUSCULAR HEMOGLOBIN (MCH)	27.1	27.0 - 32.0	pg
METHOD : CALCULATED PARAMETER	2712	2710 3210	F 5
MEAN CORPUSCULAR HEMOGLOBIN	33.2	31.5 - 34.5	g/dL
CONCENTRATION (MCHC)			
METHOD : CALCULATED PARAMETER	E.E. Laur	11.6.14.0	0/
RED CELL DISTRIBUTION WIDTH (RDW) METHOD: CALCULATED PARAMETER	5.5 Low	11.6 - 14.0	%
MENTZER INDEX	15.0		
MEAN PLATELET VOLUME (MPV)	11.1 High	6.8 - 10.9	fL
METHOD : CALCULATED PARAMETER	IIII IIIGII	0.0 - 10.9	IL
WBC DIFFERENTIAL COUNT			
NEUTROPHILS	50	40 - 80	%
METHOD: LIGHT ABSORBANCE OF CYTOCHEMICAL STAINED CELL	S IMPEDANCE		
LYMPHOCYTES	35	20 - 40	%
METHOD: LIGHT ABSORBANCE OF CYTOCHEMICAL STAINED CELL	S IMPEDANCE		

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CONSULTANT PATHOLOGIST

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METHOD: LIGHT ABSORBANCE OF CYTOCHEMICAL STAINED CELL	C IMPEDANCE		
EOSINOPHILS	5	1 - 6	%
BASOPHILS	0	0 - 2	%
METHOD: LIGHT ABSORBANCE OF CYTOCHEMICAL STAINED CELL	-	5 –	
ABSOLUTE NEUTROPHIL COUNT	2.36	2.0 - 7.0	thou/μL
ABSOLUTE LYMPHOCYTE COUNT	1.65	1 - 3	thou/µL
ABSOLUTE MONOCYTE COUNT	0.47	0.20 - 1.00	thou/µL
METHOD: CALCULATED PARAMETER			
ABSOLUTE EOSINOPHIL COUNT	0.24	0.02 - 0.50	thou/µL
ABSOLUTE BASOPHIL COUNT	0.00 Low	0.02 - 0.10	thou/μL
NEUTROPHIL LYMPHOCYTE RATIO (NLR)	1.4		

METHOD: CALCULATED PARAMETER

Interpretation(s)

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

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

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

diagnosing a case of beta thalassaemia trait.

WBC DIFFERENTIAL COUNT-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 = 3.0 years old and N

This ratio element is a calculated parameter and out of NABL scope.

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HAEMATOLOGY

MEDI WHEEL FULL BODY HEALTH CHECK UP ABOVE 40 MALE

ERYTHROCYTE SEDIMENTATION RATE (ESR), EDTA BLOOD

E.S.R 0 - 14mm at 1 hr 11

METHOD: MODIFIED WESTERGREN

GLYCOSYLATED HEMOGLOBIN(HBA1C), EDTA WHOLE **BLOOD**

Non-diabetic Adult < 5.7 HBA1C 5.4 %

Pre-diabetes 5.7 - 6.4

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

(ADA Guideline 2021)

ESTIMATED AVERAGE GLUCOSE(EAG) 108.3 < 116.0 mg/dL

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)

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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.

- 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.
- 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 CHECK UP ABOVE 40 MALE

ABO GROUP & RH TYPE, EDTA WHOLE BLOOD

TYPE O **ABO GROUP**

METHOD : SLIDE AGGLUTINATION

RH TYPE **POSITIVE**

METHOD: SLIDE AGGLUTINATION

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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BIOCHEMISTRY

MEDI WHEEL FULL BODY HEALTH CHECK UP ABOVE 40 MALE

GLUCOSE FASTING, FLUORIDE PLASMA

FBS (FASTING BLOOD SUGAR)

METHOD: HEXOKINASE

107 High

74 - 106

mg/dL

GLUCOSE, POST-PRANDIAL, PLASMA

PPBS(POST PRANDIAL BLOOD SUGAR)

128

Non-Diabetes

mg/dL

70 - 140

METHOD: HEXOKINASE

LIPID PROFILE WITH CALCULATED LDL

CHOLESTEROL, TOTAL

246 High

< 200 Desirable

mg/dL

200 - 239 Borderline High >/= 240 High

METHOD: CHOLESTEROL OXIDASE, ESTERASE, PEROXIDASE

TRIGLYCERIDES

203 High

< 150 Normal

mg/dL

150 - 199 Borderline High 200 - 499 High

>/= 500 Very High

METHOD: ENZYMATIC ASSAY

METHOD : DIRECT MEASURE - PEG CHOLESTEROL LDL

HDL CHOLESTEROL

45

< 40 Low

mg/dL

mg/dL

>/=60 High

160 High < 100 Optimal

100 - 129

Near or above optimal

130 - 159 Borderline High 160 - 189

High >/= 190 Very High

 ${\tt METHOD: CHOLESTEROL\ OXIDASE,\ ESTERASE, PEROXIDASE}$

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NON HDL CHOLESTEROL	45	Desirable: Less than 130 mg/dL Above Desirable: 130 - 159 Borderline High: 160 - 189 High: 190 - 219 Very high: > or = 220
METHOD: CALCULATED PARAMETER		
VERY LOW DENSITY LIPOPROTEIN	40.6 High	Desirable value : mg/dL 10 - 35
METHOD: CALCULATED PARAMETER		
CHOL/HDL RATIO	5.5 High	3.3-4.4 Low Risk 4.5-7.0 Average Risk 7.1-11.0 Moderate Risk > 11.0 High Risk
METHOD: CALCULATED PARAMETER		-
LDL/HDL RATIO	3.6 High	0.5 - 3.0 Desirable/Low Risk 3.1 - 6.0 Borderline/Moderate Risk >6.0 High Risk
METHOD: CALCULATED PARAMETER		

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 1	najor risk factors or evidence of end organ damage 3.		
	Familial Homozygous Hypercholesterolemi	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 (Ath	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				
Family history of premature ASCVD 4. High blood pressure				
5. Low HDL				

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

tewer treatment goals and statin initiation thresholds based on the risk categories proposed by 1211 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 < OR = 30)	< 80 (Optional goal <or 60)<="" =="" td=""><td>>OR = 50</td><td>>OR = 80</td></or>	>OR = 50	>OR = 80
Extreme Risk Group Category B	<or 30<="" =="" td=""><td><or 60<="" =="" td=""><td>> 30</td><td>>60</td></or></td></or>	<or 60<="" =="" td=""><td>> 30</td><td>>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

^{*}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	0.33	UPTO 1.2	mg/dL
METHOD: DIAZONIUM ION, BLANKED (ROCHE)			
BILIRUBIN, DIRECT	0.10	0.00 - 0.30	mg/dL
METHOD: DIAZOTIZATION			
BILIRUBIN, INDIRECT	0.23	0.00 - 0.60	mg/dL
METHOD: CALCULATED PARAMETER			
TOTAL PROTEIN	7.5	6.6 - 8.7	g/dL
METHOD: BIURET			
ALBUMIN	5.0 High	3.97 - 4.94	g/dL
METHOD: BROMOCRESOL GREEN			
GLOBULIN	2.5	2.0 - 4.0 Neonates - Pre Mature: 0.29 - 1.04	g/dL
METHOD: CALCULATED PARAMETER			
ALBUMIN/GLOBULIN RATIO	2.0	1.0 - 2.0	RATIO
METHOD: CALCULATED PARAMETER			
ASPARTATE AMINOTRANSFERASE(AST/SGOT)	26	0 - 40	U/L
ALANINE AMINOTRANSFERASE (ALT/SGPT)	38	0 - 41	U/L
METHOD: UV WITHOUT PYRIDOXAL-5 PHOSPHATE			
ALKALINE PHOSPHATASE	129	40 - 129	U/L
METHOD: PNPP - AMP BUFFER			
GAMMA GLUTAMYL TRANSFERASE (GGT)	25	8 - 61	U/L
METHOD: GAMMA GLUTAMYLCARBOXY 4NITROANILIDE			
LACTATE DEHYDROGENASE	159	135 - 225	U/L
METHOD: LACTATE -PYRUVATE			

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BLOOD UREA NITROGEN (BUN), SERUM

BLOOD UREA NITROGEN 10 6 - 20 mg/dL

METHOD: UREASE - UV

CREATININE, SERUM

CREATININE 0.87 0.70 - 1.20 mg/dL

METHOD: ALKALINE PICRATE-KINETIC

BUN/CREAT RATIO

BUN/CREAT RATIO 11.49 5.00 - 15.00

 ${\tt METHOD}: {\tt CALCULATED} \; {\tt PARAMETER} \;$

URIC ACID, SERUM

URIC ACID **7.4 High** 3.4 - 7.0 mg/dL

METHOD: URICASE, COLORIMETRIC

TOTAL PROTEIN, SERUM

TOTAL PROTEIN 7.5 6.6 - 8.7 g/dL

METHOD : BIURET

ALBUMIN, SERUM

ALBUMIN **5.0 High** 3.97 - 4.94 g/dL

METHOD: BROMOCRESOL GREEN

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GLOBULIN			
GLOBULIN	2.5	2.0 - 4.0 Neonates - Pre Mature: 0.29 - 1.04	g/dL
METHOD : CALCULATED PARAMETER			
ELECTROLYTES (NA/K/CL), SERUM			
SODIUM, SERUM METHOD: ISE INDIRECT	139	136 - 145	mmol/L
POTASSIUM, SERUM METHOD: ISE INDIRECT	4.8	3.5 - 5.1	mmol/L
CHLORIDE, SERUM	102	98 - 107	mmol/L

Interpretation(s)

METHOD: ISE INDIRECT

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 o 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.

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PATIENT ID : RANDM05128180 DRAWN

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

Interferences: Severe linemia 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

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.

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.

Chardni Garg

Dr. Pranjali Vasisht

LAB HEAD

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View Report



Agilus Diagnostics Ltd. 24 Sco, Sector 11 D Chandigarh, 160011 Punjab, India







PATIENT NAME: RANDEEP CHAWLA REF. DOCTOR: SELF

CODE/NAME & ADDRESS: C000138383

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

DELHI

NEW DELHI 110030 8800465156 ACCESSION NO : 0080XA008665

PATIENT ID : RANDM05128180

CLIENT PATIENT ID: ABHA NO : 7D 4 14/14 1

AGE/SEX

RECEIVED : 27/01/2024 09:07:27 REPORTED : 27/01/2024 14:27:30

:42 Years

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

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.

Chardni Garg

DR.CHANDNI GARG
CONSULTANT PATHOLOGIST

Coracult

Dr.Pranjali Vasisht LAB HEAD





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Agilus Diagnostics Ltd. 24 Sco, Sector 11 D Chandigarh, 160011 Punjab, India







CODE/NAME & ADDRESS: C000138383 ARCOFEMI HEALTHCARE LTD (MEDIWHEEL F-703, LADO SARAI, MEHRAULISOUTH WEST

DELHI

NEW DELHI 110030

8800465156

REF. DOCTOR: SELF

: RANDM05128180

ACCESSION NO: 0080XA008665 AGE/SEX :42 Years Male

RECEIVED: 27/01/2024 09:07:27

CLIENT PATIENT ID: ABHA NO

REPORTED :27/01/2024 14:27:30

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

PATIENT ID

CLINICAL PATH - URINALYSIS

MEDI WHEEL FULL BODY HEALTH CHECK UP ABOVE 40 MALE

PHYSICAL EXAMINATION, URINE

COLOR PALE YELLOW

APPEARANCE CLEAR

CHEMICAL EXAMINATION, URINE

PH 6.0 4.7 - 7.5

METHOD: REFLECTANCE SPECTROPHOTOMETRY- DOUBLE INDICATOR METHOD 1.003 - 1.035 SPECIFIC GRAVITY 1.030

METHOD: REFLECTANCE SPECTROPHOTOMETRY (PKA CHANGE OF PRETREATED POLY ELECTROLYTES)

NOT DETECTED **PROTEIN** NOT DETECTED

METHOD: REFLECTANCE SPECTROPHOTOMETRY (PROTEIN-ERROR-OF-INDICATORS PRINCIPLE) NOT DETECTED NOT DETECTED GLUCOSE

METHOD: REFLECTANCE SPECTROPHOTOMETRY(GLUCOSE OXIDAE/PEROXIDASE METHOD) NOT DETECTED KFTONES NOT DETECTED

METHOD: REFLECTANCE SPECTROPHOTOMETRY (SODIUM NITROPRUSSIDE REACTION)

NOT DETECTED BLOOD NOT DETECTED

METHOD: REFLECTANCE SPECTROPHOTOMETRY (PEROXIDASE METHOD)

BILIRUBIN NOT DETECTED NOT DETECTED

METHOD: REFLECTANCE SPECTROPHOTOMETRY (DIAZO REACTION)

UROBILINOGEN **NORMAL NORMAL**

METHOD: REFLECTANCE SPECTROPHOTOMETRY - EHRLICH REACTION

NITRITE NOT DETECTED NOT DETECTED

METHOD: REFLECTANCE SPECTROPHOTOMETRY, CONVERSION OF NITRATE TO NITRITE

LEUKOCYTE ESTERASE NOT DETECTED NOT DETECTED

MICROSCOPIC EXAMINATION, URINE

/HPF **NOT DETECTED** RED BLOOD CELLS NOT DETECTED

METHOD: MICROSCOPIC EXAMINATION

PUS CELL (WBC'S) 2-3 0 - 5/HPF

METHOD: MICROSCOPIC EXAMINATION

0-1 0-5 /HPF EPITHELIAL CELLS

METHOD: MICROSCOPIC EXAMINATION

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LAB HEAD







Agilus Diagnostics Ltd. 24 Sco, Sector 11 D Chandigarh, 160011 Punjab, India







PATIENT NAME: RANDEEP CHAWLA REF. DOCTOR: SELF

 CODE/NAME & ADDRESS : C000138383
 ACCESSION NO : 0080XA008665
 AGE/SEX : 42 Years
 Male

ARCOFEMI HEALTHCARE LTD (MEDIWHEEL | PATIENT ID : RANDM05128180 | DRAWN :

F-703, LADO SARAI, MEHRAULISOUTH WEST CLIENT PATIENT ID: RECEIVED : 27/01/2024 09:07:27

DELHI

NEW DELHI 110030

ABHA NO:

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

CASTS NOT DETECTED
CRYSTALS NOT DETECTED

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

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Agilus Diagnostics Ltd. 24 Sco, Sector 11 D Chandigarh, 160011 Punjab, India







PATIENT NAME: RANDEEP CHAWLA REF. DOCTOR: SELF

CODE/NAME & ADDRESS: C000138383

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

DELHÍ

NEW DELHI 110030 8800465156 ACCESSION NO: 0080XA008665

PATIENT ID : RANDM05128180

CLIENT PATIENT ID: ABHA NO : AGE/SEX :42 Years

27/01/2024 00:0

RECEIVED : 27/01/2024 09:07:27 REPORTED : 27/01/2024 14:27:30

Test Report Status Final Results Biological Reference Interval Units

Bacteria Urinary infectionwhen present in significant numbers & with pus cells.

Trichomonas vaginalis Vaginitis, cervicitis or salpingitis

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Agilus Diagnostics Ltd. 24 Sco, Sector 11 D Chandigarh, 160011 Punjab, India







CODE/NAME & ADDRESS: C000138383

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

DELHI

NEW DELHI 110030 8800465156 **REF. DOCTOR:** SELF

ACCESSION NO: **0080XA008665** AGE/SEX: 42 Years

PATIENT ID : RANDM05128180 DRAW

CLIENT PATIENT ID: ABHA NO : DRAWN

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CLINICAL PATH - STOOL ANALYSIS

MEDI WHEEL FULL BODY HEALTH CHECK UP ABOVE 40 MALE

PHYSICAL EXAMINATION, STOOL

COLOUR SAMPLE NOT RECEIVED

Dr.Ruby Jain Locum Microbilogist Dr. Nidhi Garg Lab Consultant Page 16 Of 18





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Agilus Diagnostics Ltd. 24 Sco, Sector 11 D Chandigarh, 160011 Punjab, India







PATIENT NAME: RANDEEP CHAWLA REF. DOCTOR: SELF

CODE/NAME & ADDRESS: C000138383 ACCESSION NO: 0080XA008665 AGE/SEX ARCOFEMI HEALTHCARE LTD (MEDIWHEEL

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NEW DELHI 110030

8800465156

PATIENT ID

CLIENT PATIENT ID:

: RANDM05128180

RECEIVED: 27/01/2024 09:07:27 REPORTED :27/01/2024 14:27:30

:42 Years

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

ABHA NO

SPECIALISED CHEMISTRY - HORMONE

MEDI WHEEL FULL BODY HEALTH CHECK UP ABOVE 40 MALE

THYROID PANEL, SERUM

METHOD: SANDWICH (ECLIA)

Т3	89.66	80.00 - 200.00	ng/dL
METHOD : COMPETITIVE (ECLIA) T4	6.06	5.10 - 14.10	μg/dL
METHOD: COMPETITIVE (ECLIA) TSH (ULTRASENSITIVE)	2.360	0.270 - 4.200	μIU/mL

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 hypothyroidism, 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, Free T4 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

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Agilus Diagnostics Ltd. 24 Sco, Sector 11 D Chandigarh, 160011 Punjab, India

Tel: 9111591115, Fax: CIN - U74899PB1995PLC045956







REF. DOCTOR: SELF PATIENT NAME: RANDEEP CHAWLA

CODE/NAME & ADDRESS: C000138383 ARCOFEMI HEALTHCARE LTD (MEDIWHEEL F-703, LADO SARAI, MEHRAULISOUTH WEST

DELHI

NEW DELHI 110030 8800465156

ACCESSION NO: 0080XA008665

PATIENT ID : RANDM05128180

CLIENT PATIENT ID: ABHA NO

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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

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

- 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

Fortis Hospital, Sector 62, Phase VIII, Mohali 160062

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(Poraralit

Dr. Pranjali Vasisht LAB HEAD

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Tel: 9111591115, Fax: CIN - U74899PB1995PLC045956

