



MC-2261

PATIENT NAME : RAJESH KUMAR		REF. DOCTOR : SELF	
CODE/NAME & ADDRESS : C000138361 ACROFEMI HEALTHCARE LTD (MEDIWHEEL) F-703, LADO SARAI, MEHRAULISOUTH WEST DELHI NEW DELHI 110030 8800465156		ACCESSION NO : 0028WE000272	AGE/SEX : 40 Years Male
		PATIENT ID : RAJEM10058328A	DRAWN :
		CLIENT PATIENT ID :	RECEIVED : 13/05/2023 08:47:25
		ABHA NO :	REPORTED : 17/05/2023 12:26:03

Test Report Status	Final	Results	Biological Reference Interval	Units
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HAEMATOLOGY - CBC

MEDI WHEEL FULL BODY HEALTH CHECK UP BELOW 40 MALE

BLOOD COUNTS,EDTA WHOLE BLOOD

HEMOGLOBIN (HB) METHOD : SPECTROPHOTOMETRY	13.8	13.0 - 17.0	g/dL
RED BLOOD CELL (RBC) COUNT METHOD : ELECTRICAL IMPEDANCE	4.95	4.5 - 5.5	mil/ μ L
WHITE BLOOD CELL (WBC) COUNT METHOD : ELECTRICAL IMPEDANCE	7.20	4.0 - 10.0	thou/ μ L
PLATELET COUNT METHOD : ELECTRICAL IMPEDANCE	120 Low	150 - 410	thou/ μ L

RBC AND PLATELET INDICES

HEMATOCRIT (PCV) METHOD : CALCULATED PARAMETER	42.6	40.0 - 50.0	%
MEAN CORPUSCULAR VOLUME (MCV) METHOD : DERIVED/COULTER PRINCIPLE	86.2	83.0 - 101.0	fL
MEAN CORPUSCULAR HEMOGLOBIN (MCH) METHOD : CALCULATED PARAMETER	27.9	27.0 - 32.0	pg
MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION (MCHC) METHOD : CALCULATED PARAMETER	32.4	31.5 - 34.5	g/dL
RED CELL DISTRIBUTION WIDTH (RDW) METHOD : DERIVED/COULTER PRINCIPLE	16.7 High	11.6 - 14.0	%
MENTZER INDEX METHOD : CALCULATED PARAMETER	17.4		
MEAN PLATELET VOLUME (MPV) METHOD : DERIVED/COULTER PRINCIPLE	12.6 High	6.8 - 10.9	fL

WBC DIFFERENTIAL COUNT

NEUTROPHILS METHOD : VCS TECHNOLOGY/ MICROSCOPY	59	40 - 80	%
LYMPHOCYTES METHOD : VCS TECHNOLOGY/ MICROSCOPY	29	20 - 40	%

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Patient Ref. No. 775000003205597



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MONOCYTES		9	2.0 - 10.0	%
METHOD : VCS TECHNOLOGY/ MICROSCOPY				
EOSINOPHILS		3	1.0 - 6.0	%
METHOD : VCS TECHNOLOGY/ MICROSCOPY				
BASOPHILS		0	0 - 1	%
METHOD : VCS TECHNOLOGY/ MICROSCOPY				
ABSOLUTE NEUTROPHIL COUNT		4.20	2.0 - 7.0	thou/ μ L
METHOD : CALCULATED PARAMETER				
ABSOLUTE LYMPHOCYTE COUNT		2.00	1.0 - 3.0	thou/ μ L
METHOD : CALCULATED PARAMETER				
ABSOLUTE MONOCYTE COUNT		0.70	0.2 - 1.0	thou/ μ L
METHOD : CALCULATED PARAMETER				
ABSOLUTE EOSINOPHIL COUNT		0.22	0.02 - 0.50	thou/ μ L
METHOD : CALCULATED PARAMETER				
ABSOLUTE BASOPHIL COUNT		0.00 Low	0.02 - 0.10	thou/ μ L
METHOD : CALCULATED PARAMETER				
NEUTROPHIL LYMPHOCYTE RATIO (NLR)		2.1		
METHOD : CALCULATED PARAMETER				

MORPHOLOGY**REMARKS**

THE PLATELET COUNT HAS BEEN PERFORMED BY VISUAL ASSESSMENT OF THE PERIPHERAL BLOOD SMEAR DUE TO THE PRESENCE OF GIANT PLATELETS EACH PLATELET /FIELD UNDER OIL IMMERSION (100X) WAS TAKEN TO REPRESENT 10,000 PLATELETS /MICROLITRE OF BLOOD. REFERENCE: WINTROBE'S CLINICAL HEMATOLOGY, 11TH EDITION (2004).

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 < 3.3, 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 CHECK UP BELOW 40 MALE

ERYTHROCYTE SEDIMENTATION RATE (ESR),WHOLE BLOOD

E.S.R	7	< 15	mm at 1 hr
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METHOD : MODIFIED WESTERGREN METHOD BY AUTOMATED ANALYSER

Interpretation(s)

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, Vasculitides, 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: Polycythemia 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.

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ABO GROUP

TYPE B

METHOD : COLUMN AGGLUTINATION TECHNOLOGY

RH TYPE

POSITIVE

METHOD : COLUMN AGGLUTINATION TECHNOLOGY

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 BELOW 40 MALE

GLUCOSE FASTING, FLUORIDE PLASMA

FBS (FASTING BLOOD SUGAR)	90	74 - 106	mg/dL
METHOD : HEXOKINASE			

GLYCOSYLATED HEMOGLOBIN(HBA1C), EDTA WHOLE BLOOD

HBA1C	5.0	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)	96.8	< 116.0	mg/dL
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GLUCOSE, POST-PRANDIAL, PLASMA

PPBS(POST PRANDIAL BLOOD SUGAR)	80	Non-Diabetes 70 - 140	mg/dL
METHOD : HEXOKINASE			

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Comments

Causes of Low Post Prandial Blood Sugar

The causes of low blood sugar that occurs following a meal have been divided traditionally into

- (1) Alimentary
- (2) Functional and
- (3) That in diabetics and those with impaired glucose tolerance

Alimentary Hypoglycemia usually, but not necessarily, occurs in those patients who have had gastrointestinal surgery. Accelerated absorption of a glucose load leads to marked post - prandial hyperglycemia with a corresponding exaggerated insulin release thus resulting in hypoglycemia the ensuing hypoglycemia typically occurs from one and one half to three hours after eating. This pattern of glucose intolerance and a history of gastrointestinal surgery are suggestive of the diagnosis.

Functional Hypoglycemia is quite common in adults; it may be characterized by abnormally low plasma glucose and symptoms of light headedness, shakiness, diaphoresis, weakness, fatigue occurring with the modest withholding of food. Complaints suggestive of this syndrome commonly are seen in those who have emotional problems.

Post Prandial Hypoglycemia in diabetics: Patients who are diabetics or who have impaired glucose tolerance also may experience reactive hypoglycemia. In these patients, hypoglycemia occurs later than in the group with the alimentary disorder, and insulin response is delayed and exaggerated.

LIPID PROFILE, SERUM

CHOLESTEROL, TOTAL	181	< 200 Desirable 200 - 239 Borderline High >= 240 High	mg/dL
METHOD : CHOLESTEROL OXIDASE, ESTERASE, PEROXIDASE			
TRIGLYCERIDES	179 High	< 150 Normal 150 - 199 Borderline High 200 - 499 High >= 500 Very High	mg/dL
METHOD : ENZYMATIC, END POINT			
HDL CHOLESTEROL	51	< 40 Low >= 60 High	mg/dL
METHOD : DIRECT MEASURE POLYMER-POLYANION			
CHOLESTEROL LDL	94	< 100 Optimal 100 - 129 Near or above optimal 130 - 159 Borderline High 160 - 189 High >= 190 Very High	mg/dL

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NON HDL CHOLESTEROL		130	Desirable: Less than 130 Above Desirable: 130 - 159 Borderline High: 160 - 189 High: 190 - 219 Very high: > or = 220	mg/dL
METHOD : CALCULATED PARAMETER				
VERY LOW DENSITY LIPOPROTEIN		35.8 High	Desirable value :	mg/dL
CHOL/HDL RATIO		3.6	10 - 35 3.3-4.4 Low Risk 4.5-7.0 Average Risk 7.1-11.0 Moderate Risk > 11.0 High Risk	
LDL/HDL RATIO		1.8	0.5 - 3.0 Desirable/Low Risk 3.1 - 6.0 Borderline/Moderate Risk Risk >6.0 High Risk	

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 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 major risk factors or evidence of end organ damage 3. Familial Homozygous Hypercholesterolemia
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 Therapy
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	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)	>OR = 50	>OR = 80
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
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 METHOD : DIAZONIUM ION, BLANKED (ROCHE)	0.82	UPTO 1.2	mg/dL
BILIRUBIN, DIRECT METHOD : DIAZOTIZATION	0.28	0.00 - 0.30	mg/dL
BILIRUBIN, INDIRECT METHOD : CALCULATED PARAMETER	0.54	0.00 - 0.60	mg/dL
TOTAL PROTEIN METHOD : BIURET,SERUM BLANK,ENDPOINT	7.4	6.6 - 8.7	g/dL
ALBUMIN METHOD : BROMOCRESOL GREEN	4.8	3.97 - 4.94	g/dL
GLOBULIN METHOD : CALCULATED PARAMETER	2.6	2.0 - 4.0 Neonates - Pre Mature: 0.29 - 1.04	g/dL
ALBUMIN/GLOBULIN RATIO METHOD : CALCULATED PARAMETER	1.9	1.0 - 2.0	RATIO
ASPARTATE AMINOTRANSFERASE(AST/SGOT) METHOD : UV WITHOUT P5P	24	0 - 40	U/L
ALANINE AMINOTRANSFERASE (ALT/SGPT) METHOD : UV WITHOUT P5P	29	0 - 41	U/L
ALKALINE PHOSPHATASE METHOD : PNPP, AMP BUFFER-IFCC	136 High	40 - 129	U/L
GAMMA GLUTAMYL TRANSFERASE (GGT) METHOD : G-GLUTAMYL-CARBOXY-NITROANILIDE-IFCC	12	8 - 61	U/L
LACTATE DEHYDROGENASE METHOD : L TO P, IFCC	200	135 - 225	U/L

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BLOOD UREA NITROGEN (BUN), SERUM

BLOOD UREA NITROGEN 11 6 - 20 mg/dL
METHOD : UREASE - UV

CREATININE, SERUM

CREATININE 0.97 0.70 - 1.20 mg/dL
METHOD : ALKALINE PICRATE-KINETIC

BUN/CREAT RATIO

BUN/CREAT RATIO 11.34 5.00 - 15.00
METHOD : CALCULATED PARAMETER

URIC ACID, SERUM

URIC ACID 6.7 3.4 - 7.0 mg/dL
METHOD : URICASE, COLORIMETRIC

TOTAL PROTEIN, SERUM

TOTAL PROTEIN 7.4 6.6 - 8.7 g/dL
METHOD : BIURET,SERUM BLANK,ENDPOINT

ALBUMIN, SERUM

ALBUMIN 4.8 3.97 - 4.94 g/dL
METHOD : BROMOCRESOL GREEN

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GLOBULIN

GLOBULIN	2.6	2.0 - 4.0 Neonates - Pre Mature: 0.29 - 1.04	g/dL
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METHOD : CALCULATED PARAMETER

ELECTROLYTES (NA/K/CL), SERUM

SODIUM, SERUM	141	136 - 145	mmol/L
METHOD : ISE INDIRECT			
POTASSIUM, SERUM	4.22	3.5 - 5.1	mmol/L
METHOD : ISE INDIRECT			
CHLORIDE, SERUM	102	98 - 107	mmol/L
METHOD : ISE 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, antidepressants (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, adrenal insufficiency, hyperaldosteronism, metabolic alkalosis. Drugs: chronic laxative, corticosteroids, diuretics.
Increased in: Dehydration (excessive sweating, severe vomiting or diarrhea), diabetes mellitus, diabetes insipidus, 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, high-dose 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.

Dr. Shyla Goel, M.B.B.S ,DCP
Sr.Pathologist



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Patient Ref. No. 775000003205597



PATIENT NAME : RAJESH KUMAR		REF. DOCTOR : SELF	
CODE/NAME & ADDRESS : C000138361		ACCESSION NO : 0028WE000272	AGE/SEX : 40 Years Male
ACROFEMI HEALTHCARE LTD (MEDIWHEEL)		PATIENT ID : RAJEM10058328A	DRAWN :
F-703, LADO SARAI, MEHRAULISOUTH WEST DELHI		CLIENT PATIENT ID:	RECEIVED : 13/05/2023 08:47:25
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<p>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.</p>	<p>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.</p>	<p>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)</p>
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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 so that 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, malignancy (adrenocortical, stomach, fibrosarcoma), infant of a diabetic mother, enzyme deficiency diseases (e.g. galactosemia), Drugs-insulin, ethanol, propranolol, sulfonureas, 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 Glycosuria, Glycaemic index & response to food consumed, Alimentary Hypoglycemia, Increased insulin response & sensitivity etc.

GLYCOSYLATED HEMOGLOBIN (HBA1C), EDTA WHOLE BLOOD-Used For:

- Evaluating the long-term control of blood glucose concentrations in diabetic patients.
- Diagnosing diabetes.
- Identifying patients at increased risk for diabetes (prediabetes).

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

- eAG (Estimated average glucose) converts percentage HbA1c to mg/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 :

- 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.
- Vitamin C & E are reported to falsely lower test results (possibly by inhibiting glycation of hemoglobin).
- 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.
- 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 platform (Boronate affinity chromatography) is recommended for testing of HbA1c. Abnormal Hemoglobin electrophoresis (HPLC method) is recommended for detecting a hemoglobinopathy

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 Glycosuria, 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,

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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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CLINICAL PATH - URINALYSIS

MEDI WHEEL FULL BODY HEALTH CHECK UP BELOW 40 MALE

PHYSICAL EXAMINATION, URINE

COLOR	PALE YELLOW
METHOD : VISUAL	
APPEARANCE	CLEAR
METHOD : VISUAL	

CHEMICAL EXAMINATION, URINE

PH	6.0	4.7 - 7.5
METHOD : DOUBLE INDICATOR PRINCIPLE		
SPECIFIC GRAVITY	1.020	1.003 - 1.035
METHOD : PKA CHANGE OF PRETREATED POLYELECTROLYTES		
PROTEIN	NOT DETECTED	NOT DETECTED
METHOD : PROTEIN- ERROR INDICATOR		
GLUCOSE	NOT DETECTED	NOT DETECTED
METHOD : OXIDASE-PEROXIDASE REACTION		
KETONES	NOT DETECTED	NOT DETECTED
METHOD : ACETOACETIC REACTION WITH NITROPRUSSIDE		
BLOOD	NOT DETECTED	NOT DETECTED
METHOD : PEROXIDASE-LIKE ACTIVITY OF HEMOGLOBIN		
BILIRUBIN	NOT DETECTED	NOT DETECTED
METHOD : DIAZOTIZATION		
UROBILINOGEN	NORMAL	NORMAL
METHOD : MODIFIED EHRlich REACTION		
NITRITE	NOT DETECTED	NOT DETECTED
METHOD : CONVERSION OF NITRATE TO NITRITE		
LEUKOCYTE ESTERASE	NOT DETECTED	NOT DETECTED
METHOD : ESTERASE HYDROLYSIS ACTIVITY		

MICROSCOPIC EXAMINATION, URINE

RED BLOOD CELLS	NOT DETECTED	NOT DETECTED	/HPF
METHOD : MICROSCOPIC EXAMINATION			
PUS CELL (WBC'S)	1-2	0-5	/HPF

Dr. Neena Verma
Senior Pathologist



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		PATIENT ID : RAJEM10058328A	DRAWN :
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METHOD : MICROSCOPIC EXAMINATION EPITHELIAL CELLS	1-2	0-5	/HPF
METHOD : MICROSCOPIC EXAMINATION CASTS	NOT DETECTED		
METHOD : MICROSCOPIC EXAMINATION CRYSTALS	NOT DETECTED		
METHOD : MICROSCOPIC EXAMINATION BACTERIA	NOT DETECTED	NOT DETECTED	
METHOD : MICROSCOPIC EXAMINATION YEAST	NOT DETECTED	NOT DETECTED	
REMARKS	MICROSCOPIC EXAMINATION DONE ON CENTRIFUGED URINEPLEASE NOTE THAT GRADING OF BACTERIA NEEDS TO BE CORELATED WITH THE CULTURE IN CASE FOUND SIGNIFICANT CLINICALLY. OCCASIONAL BACTERIA/YEAST CELLS SEEN IN MICROSCOPY CAN BE A PART OF SURROUNDING SKIN FLORA ALSO.		

METHOD : MANUAL

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

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PATIENT ID : RAJEM10058328A

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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 infection when present in significant numbers & with pus cells.
Trichomonas vaginalis	Vaginitis, cervicitis or salpingitis

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SPECIALISED CHEMISTRY - HORMONE

MEDI WHEEL FULL BODY HEALTH CHECK UP BELOW 40 MALE

THYROID PANEL, SERUM

T3	133.0	80.00 - 200.00	ng/dL
METHOD : ECLIA			
T4	7.37	5.10 - 14.10	µg/dL
METHOD : ECLIA			
TSH (ULTRASENSITIVE)	4.590 High	0.270 - 4.200	µIU/mL
METHOD : ECLIA			

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

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CODE/NAME & ADDRESS : C000138361 ACROFEMI HEALTHCARE LTD (MEDIWHEEL) F-703, LADO SARAI, MEHRAULISOUTH WEST DELHI NEW DELHI 110030 8800465156	ACCESSION NO : 0028WE000272 PATIENT ID : RAJEM10058328A CLIENT PATIENT ID : ABHA NO :	AGE/SEX : 40 Years Male DRAWN : RECEIVED : 13/05/2023 08:47:25 REPORTED : 17/05/2023 12:26:03
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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. Guidelines of the American Thyroid association during pregnancy and Postpartum, 2011.
NOTE: It is advisable to detect Free T3, Free T4 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.

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MEDI WHEEL FULL BODY HEALTH CHECK UP BELOW 40 MALE

XRAY-CHEST

>>> BOTH THE LUNG FIELDS ARE CLEAR
 >>> BOTH THE COSTOPHRENIC AND CARIOPHRENIC ANGELS ARE CLEAR
 >>> BOTH THE HILA ARE NORMAL
 >>> CARDIAC AND AORTIC SHADOWS APPEAR NORMAL
 >>> BOTH THE DOMES OF THE DIAPHRAM ARE NORMAL
 >>> VISUALIZED BONY THORAX IS NORMAL
 IMPRESSION NORMAL

TMT OR ECHO

TMT OR ECHO TMT DONE - NORMAL

ECG

ECG WITHIN NORMAL LIMITS

MEDICAL HISTORY

RELEVANT PRESENT HISTORY CHRONIC COUGH.
 RELEVANT PAST HISTORY COVID POSITOVE APRIL 2021.
 RELEVANT PERSONAL HISTORY MARRIED,NON,VEGETARIAN.
 RELEVANT FAMILY HISTORY MOTHER-HTN.
 OCCUPATIONAL HISTORY FATHER-DIABETIC.
 HISTORY OF MEDICATIONS NOT SIGNIFICANT
 NOT SIGNIFICANT

ANTHROPOMETRIC DATA & BMI

HEIGHT IN METERS 1.70 mts



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Patient Ref. No. 775000003205597

PATIENT NAME : RAJESH KUMAR		REF. DOCTOR : SELF	
CODE/NAME & ADDRESS : C000138361 ACROFEMI HEALTHCARE LTD (MEDIWHEEL) F-703, LADO SARAI, MEHRAULISOUTH WEST DELHI NEW DELHI 110030 8800465156		ACCESSION NO : 0028WE000272 PATIENT ID : RAJEM10058328A CLIENT PATIENT ID: ABHA NO :	
		AGE/SEX : 40 Years Male DRAWN : RECEIVED : 13/05/2023 08:47:25 REPORTED : 17/05/2023 12:26:03	

Test Report Status	Final	Results	Biological Reference Interval	Units
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WEIGHT IN KGS.	72.8			Kgs
BMI	25			kg/sqmts
		BMI & Weight Status as follows Below 18.5: Underweight 18.5 - 24.9: Normal 25.0 - 29.9: Overweight 30.0 and Above: Obese		

GENERAL EXAMINATION

MENTAL / EMOTIONAL STATE	NORMAL
PHYSICAL ATTITUDE	NORMAL
GENERAL APPEARANCE / NUTRITIONAL STATUS	HEALTHY
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	83 / MIN REGULAR, ALL PERIPHERAL PULSES WELL FELT, NO CAROTID BRUIT
RESPIRATORY RATE	NORMAL

CARDIOVASCULAR SYSTEM

BP	136/79	mm/Hg
PERICARDIUM	NORMAL	
APEX BEAT	NORMAL	
HEART SOUNDS	NORMAL	
MURMURS	ABSENT	



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 New Delhi, India
 Tel : 9111591115, Fax :
 CIN - U74899PB1995PLC045956
 Email : wellness.eastdelhi@srl.in



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RESPIRATORY SYSTEM

SIZE AND SHAPE OF CHEST	NORMAL
MOVEMENTS OF CHEST	SYMMETRICAL
BREATH SOUNDS INTENSITY	NORMAL
BREATH SOUNDS QUALITY	VESICULAR (NORMAL)
ADDED SOUNDS	ABSENT

PER ABDOMEN

APPEARANCE	NORMAL
VENOUS PROMINENCE	ABSENT
LIVER	NOT PALPABLE
SPLEEN	NOT PALPABLE

CENTRAL NERVOUS SYSTEM

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

MUSCULOSKELETAL SYSTEM

SPINE	NORMAL
JOINTS	NORMAL



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 F-703, LADO SARAI, MEHRAULISOUTH WEST
 DELHI
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BASIC EYE EXAMINATION

CONJUNCTIVA	NORMAL
EYELIDS	NORMAL
EYE MOVEMENTS	NORMAL
CORNEA	NORMAL
DISTANT VISION RIGHT EYE WITHOUT GLASSES	6/12
DISTANT VISION LEFT EYE WITHOUT GLASSES	6/12
NEAR VISION RIGHT EYE WITHOUT GLASSES	NORMAL
NEAR VISION LEFT EYE WITHOUT GLASSES	NORMAL
COLOUR VISION	NORMAL

BASIC ENT EXAMINATION

EXTERNAL EAR CANAL	NORMAL
TYMPANIC MEMBRANE	NORMAL
NOSE	NO ABNORMALITY DETECTED
SINUSES	NORMAL
THROAT	NO ABNORMALITY DETECTED
TONSILS	NOT ENLARGED

BASIC DENTAL EXAMINATION

TEETH	NORMAL
GUMS	CALCULUS PRESENT.

SUMMARY

RELEVANT HISTORY	NOT SIGNIFICANT
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RELEVANT GP EXAMINATION FINDINGS	NOT SIGNIFICANT
RELEVANT LAB INVESTIGATIONS	WITHIN NORMAL LIMITS
RELEVANT NON PATHOLOGY DIAGNOSTICS	NO ABNORMALITIES DETECTED
REMARKS / RECOMMENDATIONS	"NO ABNORMALITY FOUND OUT OF THE DIAGNOSTIC PACKAGE REQUESTED. GENERAL PHYSICAL EXAMINATION IS NORMAL."



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MEDI WHEEL FULL BODY HEALTH CHECK UP BELOW 40 MALE

ULTRASOUND ABDOMEN

ULTRASOUND ABDOMEN

BORDERLINE PROSTATOMEGALY

Interpretation(s)

MEDICAL

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

****End Of Report****

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

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.

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