

CLIENT'S NAME AND ADDRESS:
ACROFEMI HEALTHCARE LTD (MEDIWHEEL)
F-703, F-703, LADO SARAI, MEHRAULI

SOUTH WEST DELHI NEW DELHI 110030 DELHI INDIA 8800465156

SRL Ltd S.K. Tower,Hari Niwas, LBS Marg THANE, 400602

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

Email: customercare.thane@srl.in

PATIENT NAME: RAM DATTARAM SURVE PATIENT ID: RAMDM060268181

ACCESSION NO: 0181VI000337 AGE: 54 Years SEX: Male

DRAWN: RECEIVED: 10/09/2022 10:36 REPORTED: 14/09/2022 13:14

REFERRING DOCTOR: SELF CLIENT PATIENT ID:

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

MEDI WHEEL FULL BODY HEALTH CHECK UP ABOVE 40 MALE

PHYSICAL EXAMINATION, URINE			
COLOR	PALE YELLOW		
METHOD: VISUAL INSPECTION			
APPEARANCE	CLEAR		
METHOD: VISUAL INSPECTION			
SPECIFIC GRAVITY	1.025	1.003 - 1.035	
METHOD: IONIC CONCENTRATION METHOD			
BLOOD COUNTS,EDTA WHOLE BLOOD			
HEMOGLOBIN	14.2	13.0 - 17.0	g/dL
METHOD: SLS-HEMOGLOBIN DETECTION METHOD			
RED BLOOD CELL COUNT	4./4	4.5 - 5.5	mil/µL
METHOD: HYDRODYNAMIC FOCUSING BY DC DETECTION			
WHILE BLOOD CELL COUNT	5.05	4.0 - 10.0	thou/µL
METHOD: FLUORESCENCE FLOW CYTOMETRY			
PLATELET COUNT	254	150 - 410	thou/µL
METHOD: HYDRODYNAMIC FOCUSING BY DC DETECTION			
RBC AND PLATELET INDICES			
HEMATOCRIT	43.2	40.0 - 50.0	%
METHOD : CUMULATIVE PULSE HEIGHT DETECTION METHOD			
MEAN CORPUSCULAR VOL	91.1	83.0 - 101.0	†L
METHOD: CALCULATED FROM RBC & HCT			
MEAN CORPUSCULAR HGB.	30.0	27.0 - 32.0	pg
METHOD: CALCULATED FROM THE RBC & HGB			
MEAN CORPUSCULAR HEMOGLOBIN	32.9	31.5 - 34.5	g/dL
CONCENTRATION METHOD: CALCULATED FROM THE HGB & HCT			
MENTZER INDEX	19.2		
RED CELL DISTRIBUTION WIDTH	12.4	11.6 - 14.0	%
METHOD : CALCULATED FROM RBC SIZE DISTRIBUTION CURVE			
MEAN PLATELET VOLUME	10.3	6.8 - 10.9	fL
METHOD : CALCULATED FROM PLATELET COUNT & PLATELET HEMA	TOCRIT		
CHEMICAL EXAMINATION, URINE			
P⊢	6.0	4.7 - 7.5	
METHOD : DOUBLE INDICATOR PRINCIPLE			
PROTEIN	DETECTED (TRACE)	NOT DETECTED	
# 		· ·	



METHOD: TETRA BROMOPHENOL BLUE/SULFOSALICYLIC ACID

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GLUCOSE	NOT DETECTED	NOT DETECTED	
METHOD : GLUCOSE OXIDASE PEROXIDASE			
KETONES	NOT DETECTED	NOT DETECTED	
METHOD: NITROPRUSSIDE REACTION			
BLOOD	NOT DETECTED	NOT DETECTED	
METHOD: PEROXIDASE	NORMAL	NORMA	
UROBILINOGEN	NORMAL	NORMAL	
METHOD: MODIFIED EHRLICH REACTION	NOT DETECTED	NOT DETECTED	
NITRITE	NOT DETECTED	NOT DETECTED	
METHOD: 1,2,3,4-TETRAHYDROBENZO(H)QUINOLIN-3-OL LEUKOCYTE ESTERASE	NOT DETECTED	NOT DETECTED	
	NOT DETECTED	NOT DETECTED	
WBC DIFFERENTIAL COUNT - NLR			
SEGMENTED NEUTROPHILS	61	40 - 80	%
METHOD: FLOW CYTOMETRY WITH LIGHT SCATTERING			
ABSOLUTE NEUTROPHIL COUNT	3.06	2.0 - 7.0	thou/µL
METHOD: FLOW CYTOMETRY WITH LIGHT SCATTERING	22	20 40	0/
LYMPHOCYTES	32	20 - 40	%
METHOD: FLOW CYTOMETRY WITH LIGHT SCATTERING ABSOLUTE LYMPHOCYTE COUNT	1.62	1.0 - 3.0	thoughd
METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING	1.02	1.0 - 3.0	thou/µL
NEUTROPHIL LYMPHOCYTE RATIC (NLR)	1.9		
` '	2	1 - 6	%
EOSINOPHILS METHOD: FLOW CYTOMETRY WITH LIGHT SCATTERING	2	1 - 0	%0
ABSOLUTE EOSINOPHIL COUNT	0.08	0.02 - 0.50	thou/µL
METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING	0.00	0.02 - 0.50	ti lou/µL
MONOCYTES	5	2 - 10	%
METHOD: FLOW CYTOMETRY WITH LIGHT SCATTERING	9	2 10	70
ABSOLUTE MONOCYTE COUNT	0.26	0.2 - 1.0	thou/µL
METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING	5.25	3.2 1.3	a roa, p.e.
DIFFERENTIAL COUNT PERFORMED ON:	EDTA SMEAR		
MICROSCOPIC EXAMINATION, URINE			
PUS CELL (WBC'S)	1-2	0-5	/HPF
METHOD: MICROSCOPIC EXAMINATION	1 2		<i>y</i> 1 11 1
EPITHELIAL CELLS	0-1	0-5	/HPF
METHOD: MICROSCOPIC EXAMINATION	_ *		,
ERYTHROCYTES (RBC'S)	NOT DETECTED	NOT DETECTED	/HPF
METHOD: MICROSCOPIC EXAMINATION	·		* * * * * * * * * * * * * * * * * * *
CASTS	NOT DETECTED		







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PATIENT ID:

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mm at 1 hr

%

mg/dL

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METHOD: MICROSCOPIC EXAMINATION		
CRYSTALS	NOT DETECTED	
METHOD: MICROSCOPIC EXAMINATION		
BACTERIA	NOT DETECTED	NOT DETECTED
METHOD: MICROSCOPIC EXAMINATION		
YEAST	NOT DETECTED	NOT DETECTED
REMARKS	PRECEINCE OF URINA	RY PROTIEN RECHECK BY MANUAL METHOD.
MORPHOLOGY		
RBC	NORMOCYTIC NORMO	OCHROMIC
WBC	NORMAL MORPHOLOG	SY
METHOD: MICROSCOPIC EXAMINATION		
PLATELETS	ADEQUATE	
ERYTHRO SEDIMENTATION RATE, BLOC	DD .	

SEDIMENTATION RATE (ESR)	06
METHOD: WESTERGREN METHOD	

GLYCOSYLATED HEMOGLOBIN, EDTA WHOLE BLOOD

Non-diabetic: < 5.7 Pre-diabetics: 5.7 - 6.4 Diabetics: > or = 6.5 ADA Target: 7.0 Action suggested: > 8.0

0 - 14

METHOD: HPLC

MEAN PLASMA GLUCOSE 116.9 High < 116.0 mg/dL

METHOD : CALCULATED PARAMETER
GLUCOSE, FASTING, PLASMA

GLUCOSE, FASTING, PLASMA 72.4 Low Normal 75 - 99

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

METHOD: ENZYMATIC REFERENCE METHOD WITH HEXOKINASE

GLUCOSE, POST-PRANDIAL, PLASMA

GLYCOSYLATED HEMOGLOBIN (HBA1C)

GLUCOSE, POST-PRANDIAL, PLASMA 72 70 - 139 mg/dL

METHOD: ENZYMATIC REFERENCE METHOD WITH HEXOKINASE

CORONARY RISK PROFILE, SERUM

CHOLESTEROL 198 Desirable cholesterol level mg/dL

< 200

Borderline high cholesterol

200 - 239 High cholesterol > / = 240

METHOD: ENZYMATIC COLORIMETRIC ASSAY







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TRIGLYCERIDES	56	Normal: < 150 Borderline high: 150 - 199 High: 200 - 499 Very High: >/= 500	mg/dL
METHOD: ENZYMATIC COLORIMETRIC ASSAY			
HDL CHOLESTEROL	39.5		mg/dL
CHOLESTEROL LDL	147		mg/dL
NON HDL CHOLESTEROL	158		mg/dL
CHOL/HDL RATIO	5.0		
LDL/HDL RATIO	3.7		
VERY LOW DENSITY LIPOPROTEIN	11.2	< OR = 30.0	mg/dL
LIVER FUNCTION PROFILE, SERUM			
BILIRUBIN, TOTAL	0.56	Upto 1.2	mg/dL
METHOD: COLORIMETRIC DIAZO			
BILIRUBIN, DIRECT	0.26	< 0.30	mg/dL
BILIRUBIN, INDIRECT	0.3	0.1 - 1.0	mg/dL
TOTAL PROTEIN METHOD: COLORIMETRIC	7.1	6.0 - 8.0	g/dL
ALBUMIN	4.6	3.97 - 4.94	g/dL
METHOD: COLORIMETRIC			
GLOBULIN	2.5	2.0 - 3.5	g/dL
ALBUMIN/GLOBULIN RATIO	1.8	1.0 - 2.1	RATIO
ASPARTATE AMINOTRANSFERASE (AST/SGOT) METHOD: UV ABSORBANCE	32	< OR = 50	U/L
ALANINE AMINOTRANSFERASE (ALT/SGPT) METHOD: UV ABSORBANCE	25	< OR = 50	U/L
ALKALINE PHOSPHATASE METHOD: COLORIMETRIC	53	40 - 129	U/L
GAMMA GLUTAMYL TRANSFERASE (GGT) METHOD: ENZYMATIC, COLORIMETRIC	10	0 - 60	U/L
LACTATE DEHYDROGENASE METHOD: UV ABSORBANCE	213	125 - 220	U/L
SERUM BLOOD UREA NITROGEN			
BLOOD UREA NITROGEN METHOD: ENZYMATIC ASSAY	9	6 - 20	mg/dL
CREATININE, SERUM			
CREATININE	0.93	0.7 - 1.2	mg/dL







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METHOD: COLORIMETRIC			
BUN/CREAT RATIO			
BUN/CREAT RATIO	9.68	8.0 - 15.0	
URIC ACID, SERUM			
URIC ACID	4.9	3.4 - 7.0	mg/dL
METHOD: ENZYMATIC COLORIMETRIC ASSAY			
TOTAL PROTEIN, SERUM			
TOTAL PROTEIN	7.1	6.0 - 8.0	g/dL
METHOD: COLORIMETRIC			
ALBUMIN, SERUM			
ALBUMIN	4.6	3.97 - 4.94	g/dL
METHOD: COLORIMETRIC			
GLOBULIN			
GLOBULIN	2.5	2.0 - 3.5	g/dL
ELECTROLYTES (NA/K/CL), SERUM			
SODIUM	138	136 - 145	mmol/L
POTASSIUM	4.76	3.5 - 5.1	mmol/L
CHLORIDE	101	98 - 107	mmol/L
THYROID PANEL, SERUM			
T3	127.0	80 - 200	ng/dL
METHOD: ELECTROCHEMILUMINESCENCE			
T4	7.97	5.1 - 14.1	µg/dL
METHOD: ELECTROCHEMILUMINESCENCE			
TSH 3RD GENERATION	1.840	0.27 - 4.2	µIU/mL
METHOD: ELECTROCHEMILUMINESCENCE			
ABO GROUP & RH TYPE, EDTA WHOLE BLOOD			
ABO GROUP	TYPE B		
METHOD: GEL COLUMN AGGLUTINATION METHOD.			
RH TYPE	NEGATIVE		
METHOD: GEL COLUMN AGGLUTINATION METHOD.			
XRAY-CHEST			
IMPRESSION	NO ABNORMALITY DETEC	IED	



TMT OR ECHO

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TMT OR ECHO 2D ECHO:-

Structurally normal valves.

No RWMA.

Good Left Ventricular systolic function. LVEF 60%

Normal LV Diastolic function. No e/o pulmonary hypertension

ECG

ECG LEFT ANTERIOR FASCICULAR BLOCK.

MEDICAL HISTORY

RELEVANT PRESENT HISTORY ON CPAP FOR OBSTRUCTIVE APRIOEA.

RELEVANT PAST HISTORY PAST H/O DEPRESSION NOT ON ANY TREATMENT AT PREASENT. RELEVANT PERSONAL HISTORY MARRIED / MIXED DIET / NO ALLERGIES / NO SMOKING / OCC

ALCOHOL.

HIGH BLOOD PRESSURE & HEART DISEASE: MOTHER. RELEVANT FAMILY HISTORY

HISTORY OF MEDICATIONS NOT SIGNIFICANT

ANTHROPOMETRIC DATA & BMI

HEIGHT IN METERS 1.68 mts WEIGHT IN KGS. 92 Kgs 33 BMI & Weight Status as follows: kg/sqmts RMI

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

GENERAL EXAMINATION

NORMAL MENTAL / EMOTIONAL STATE PHYSICAL ATTITUDE NORMAL GENERAL APPEARANCE / NUTRITIONAL STATUS OBESE 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 82/MIN.REGULAR, ALL PERIPHERAL PULSES WELL FELT, NO CAROTID

BRUIT







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DE2070 (TOD) / D (TE	Norwa	
RESPIRATORY RATE	NORMAL	
CARDIOVASCULAR SYSTEM		
BP	130/70 MM HG (SUPINE)	mm/Hg
PERICARDIUM	NORMAL	
APEX BEAT	NORMAL	
HEART SOUNDS	NORMAL	
MURMURS	ABSENT	
RESPIRATORY SYSTEM		
SIZE AND SHAPE OF CHEST	NORMAL	
MOVEMENTS OF CHEST	SYMMETRICAL	
BREATH SOUNDS INTENSITY	NORMAL	
BREATH SOUNDS QUALITY	VESICULAR (NORMAL)	
ADDED SOUNDS	ABSENT	
PER ABDOMEN		
APPEARANCE	NORMAL	
VENOUS PROMINENCE	ABSENT	
LIVER	NOT PALPABLE	
SPLEEN	NOT PALPABLE	
HERNIA	ABSENT	
CENTRAL NERVOUS SYSTEM		
HIGHER FUNCTIONS	NORMAL	
CRANIAL NERVES	NORMAL	
CEREBELLAR FUNCTIONS	NORMAL	
SENSORY SYSTEM	NORMAL	
MOTOR SYSTEM	NORMAL	
REFLEXES	NORMAL	
MUSCULOSKELETAL SYSTEM		
SPINE	NORMAL	
JOINTS	NORMAL	
BASIC EYE EXAMINATION		
CONJUNCTIVA	NORMAL	
EYELIDS	NORMAL	
EYE MOVEMENTS	NORMAL	
CORNEA	NORMAL	







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DISTANT VISION RIGHT EYE WITHOUT GLASSES REDUCED VISUAL ACUITY 6/18 DISTANT VISION LEFT EYE WITHOUT GLASSES REDUCED VISUAL ACUITY 6/18 NEAR VISION RIGHT EYE WITHOUT GLASSES REDUCED VISUAL ACUITY N/24 NEAR VISION LEFT EYE WITHOUT GLASSES REDUCED VISUAL ACUITY N/24 NEAR VISION RIGHT EYE WITH GLASSES REDUCED VISUAL ACUITY N/8 NEAR VISION LEFT EYE WITH GLASSES WITHIN NORMAL LIMIT NORMAL

COLOUR VISION

SUMMARY

RELEVANT HISTORY NOT SIGNIFICANT OBESE: BMI 33 RELEVANT GP EXAMINATION FINDINGS

REDUCED VISUAL ACUITY FOR DISTANT VISION. REMARKS / RECOMMENDATIONS

1) OPHTHALMOLOGY CONSULT FOR REDUCED VISUAL ACUITY. LOW FAT, LOW CALORIE, LOW CARBOHYDRATE, HIGH FIBRE DIET, REGULAR EXERCISE, REGULAR WALK FOR 30-40 MIN DAILY.

3) DRINK 3-4 LITTER WATER DAILY.

4) REPEAT URINE ROUTINE AFTER 15 DAYS.

5) ENT CONSULTATION FOR OBSTRUCTIVE SLEEP APNOEA.

6) REGULAR FOLLOW UP WITH PSYCHIATRIST.

7) UROLOGY CONSULT FOR PROSTATOMEGALY & RENAL CALCULAU.

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.

WBC DIFFERENTIAL COUNT - NLRThe 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. MICROSCOPIC EXAMINATION, URINE-

Routine urine analysis assists in screening and diagnosis of various metabolic, urological, kidney and liver disorders

Protein: Elevated proteins can be an early sign of kidney disease. Uninary protein excretion can also be temporarily elevated by strenuous exercise, orthostatic proteinuria, dehydration, uninary tract infections and acute illness with fever

Glucose: Uncontrolled diabetes mellitus can lead to presence of glucose in urine. Other causes include pregnancy, hormonal disturbances, liver disease and certain medications.

Ketones: Uncontrolled diabetes mellitus car lead to presence of ketones in urine. Ketones can also be seen in starvation, frequent vomiting, pregnancy and strenuous

Blood: Occult blood can occur in urine as intact erythrocytes or haemoglobin, which can occur in various urological, nephrological and bleeding disorders. Leukocytes: An increase in leukocytes is an indication of inflammation in urinary tract or kidneys. Most common cause is bacterial urinary tract intection.

Nitrite: Many bacteria give positive results when their number is high. Nitrite concentration during infection increases with length of time the urine specimen is retained in

bladder prior to collection.
pH: The kidneys play an important role in maintaining acid base balance of the body. Conditions of the body producing acidosis/ alkalosis or ingestion of certain type of food can affect the pH of urine.

Specific gravity: Specific gravity gives an indication of how concentrated the urine is. Increased specific gravity is seen in conditions like dehydration, glycosuria and proteinuria while decreased specific gravity is seen in excessive fluid intake, renal failure and diabetes insipidus.

Bilirubin: In certain liver diseases such as biliary obstruction or hepatitis, bilirubin gets excreted in urine

Urobilinogen: Positive results are seen in liver diseases like hepatitis and cirrhosis and in cases of hemolytic anemia

ERYTHRO SEDIMENTATION RATE, BLOOD-







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Erythrocyte sedimentation rate (ESR) is a non - specific phenomena and is clinically useful in the diagnosis and monitoring of disorders associated with an increased production of acute phase reactants. The ESR is increased in pregnancy from about the 3rd month and returns to normal by the 4th week post partum. ESR is influenced by age, sex, menstrual cycle and drugs (eg. corticosteroids, contraceptives). It is especially low (0-1mm) in polycythaemia, hypofibrinogenemia or congestive cardiac failure and when there are abnormalities of the redicells such as polkilocytosis, spherocytosis or sickle cells.

Reference:

- Nathan and Oski's Haematology of Infancy and Childhood, 5th edition
 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, EDTA WHOLE BLOOD-

Glycosylatec hemoglobin (GHb) has been firmly established as an index of long-term blood glucose concentrations and as a measure of the risk for the development of complications in patients with diabetes mellitus. Formation of GHb is essentially irreversible, and the concentration in the blood depends on both the life span of the reciblood cell (average 120 days) and the blood glucose concentration. Because the rate of formation of GHb is directly proportional to the concentration of glucose in the blood, the GHb concentration represents the integrated values for glucose over the preceding 6-8 weeks.

Any condition that alters the life span of the red blood cells has the potential to alter the GHb level. Samples from patients with hemolytic anemias will exhibit decreased

glycated hem oglobin values due to the shortened life span of the red cells. This effect will depend upon the severity of the anemia. Samples from patients with polycythemia or post-splenectomy may exhibit increase glycated hemoglobin values due to a somewhat longer life span of the rec cells.

Glycosylatec hemoglobins results from patients with HbSS, HbCC, and HbSC and HbBC and the must be interpreted with caution, given the pathological processes, including anemia, increased rec cell turnover, transfusion requirements, that adversely impact HbA1c as a marker of long-term glycemic control. In these conditions, alternative forms of testing such as glycated serum protein (fructosamine) should be considered.

Targets should be individualized; More or less stringent glycemic goals may be appropriate for individual patients. Goals should be individualized based on duration of diabetes, age/life expectancy, comorbid conditions, known CVD or advanced microvascular complications, hypoglycemia unawareness, and individual patient considerations.

References

- 1. Tietz Textbook of Clinical Chemistry and Molecular Diagnostics, edited by Carl A Burtis, Edward R. Ashwood, David E Bruns, 4th Edition, Elsevier publication, 2006,
- 2. Forsham PH. Diabetes Mellitus: A rational plan for management. Postgrad Med 1982, 71,139-154.
- 3. Mayer TK, Freedman ZR: Protein glycosylation in Diabetes Mellitus: A review of laboratory measurements and their clinical utility. Clin Chim Acta 1983, 127, 147-184. GLUCOSE, FASTING, PLASMA-

ADA 2021 guidelines for adults, after 8 hrs fasting is as follows: Pre-diabetics: 100 - 125 mg/dL

Diabetic: > or = 126 mg/dL
Diabetic: > or = 126 mg/dL
GLUCOSE, POST-PRANDIAL, PLASMA-ADA Guidelines for 2hr post prandial glucose levels is only after ingestion of 75grams of glucose in 300 ml water, over a period of 5

LIVER FUNCTION PROFILE, SERUM-LIVER FUNCTION PROFILE

Bilirubin is a yellowish pigment founc in bile and is a breakdowr 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 termec 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, Paget's disease, Rickets, Sarcoidosis etc. Lower-than-normal ALP levels seen in Hypophosphatasia, Malnutrition, Protein deficiency, Wilson's disease. GGT is an enzyme found in cell membranes of many tissues mainly in the liver, kidney and pancreas. It is also found in other tissues including intestine, spleen, heart, brain and seminal vesicles. The highest concentration is in the kidney, but the liver is considered the source of normal enzyme activity. Serum GGT has been widely used as an index of liver dystunction. Elevated serum GGT activity can be found in diseases of the liver, billiary system. normal enzyme activity. Serum GG1 has been widely used as an index of liver dystruction. Elevated Serum GG1 activity can be found in diseases of the liver, billary system and paneraes. Conditions that increase serum GG1 are obstructive liver disease, high alcohol consumptor and use of enzyme-inducing drugs etc. Serum total protein, also known as total protein, is a biochemical test for measuring the total amount of protein in serum. Protein in the plasma is made up of albumir and globulin. Higher-than-normal levels may be due to: Chronic inflammation or infection, including HIV and hepatitis B or C, Multiple myeloma, Waldenstrom's disease. Lower-than-normal levels may be due to: Agammaglobulinemia, Bleeding (hemorrhage), Burns, Glomerulonephritists, Liver disease, Malabsorption, Malnutrition, Nephrotic syndrome, Protein in human blood plasma. It is produced in the liver. Albumir 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 SERUM BLOOD UREA NITRÓGEN-

Causes of Increased levels

Pre renal

- High protein diet, Increasec protein catabolism, GI haemorrhage, Cortisol, Dehydration, CHF Renal
- Renal Failure



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PATIENT ID:

RAMDM060268181

Email: customercare.thane@srl.in

PATIENT NAME: RAM DATTARAM SURVE

ACCESSION NO: 0181VI000337 AGE: 54 Years SEX: Male

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

Post Renal

Malignancy, Nephrolithiasis, Prostatism

Causes of decreased levels

- 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
- Muscular dystrophy

URIC ACID, SERUM-

Causes of Increased levels

- Dietary

 High Protein Intake. Prolonged Fasting.
- Rapid weight loss.

Gout

Lesch nyhan syndrome.

Type 2 ĎM.

Metabolic syndrome.

Causes of decreasec levels
• Low Zinc Intake

- · Multiple Sclerosis

Nutritional tips to manage increased Uric acid levels
• Drink plenty of fluids

- Limit animal proteins
- · High Fibre foods
- Vit C Intake
- Antioxidant rich foods

TOTAL PROTEIN, SERUM-

Serum total protein, also known as total protein, is a biochemical test for measuring the total amount of protein in serum. Protein in the plasma is made up of albumin and

Higher-than-normal levels may be due to: Chronic inflammation or infection, including HIV and hepatitis B or C, Multiple myeloma, Waldenstrom's disease Lower-than-normal levels may be due to: Agammaglobulinemia, Bleeding (hemorrhage), Burns, Glomerulonephritis, Liver disease, Malabsorption, Malnutrition, Nephrotic syndrome, Protein-Tosing 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.

ELECTROLYTES (NA/K/CL), SERUMSodium levels are Increased in dehydration, cushing's syndrome, aldosteronism & decreased in Addison's disease, hypopituitarism, liver disease. Hypokalemia (low K) is common in vomiting, diarrhea, alcoholism, folic acid deficiency and primary aldosteronism. Hyperkalemia may be seen in end-stage renal failure, hemolysis, trauma, Addison's disease, metabolic acidosis, acute starvation, dehydration, and with rapid K infusion. Chloride is increased in dehydration, renal tubular acidosis (hyperchloremia metabolic acidosis), acute renal failure, metabolic acidosis associated with prolonged diarrhea and loss of sodium bicarbonate, diabetes insipidus, adrenocortical hyperfuction, salicylate intoxication and with excessive infusion of isotonic saline or extremely high dietary intake of salt. Chloride is decreased in overhydration, chronic

hypertuction, salicylate intoxication and with excessive infusion of isotonic saline or extremely high dietary intake of salt. Chloride is decreased in overhydration, chronic respiratory acidosis, salt-losing nephritis, metabolic alkalosis, congestive heart failure, Addisonian crisis, certain types of metabolic acidosis, persistent gastric secretion and prolonged vomiting,
THYROID PANEL, SERUMTriiodothyronine T3, is a thyroid hormone. It affects 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.
Thyroxine T4, Thyroxine's principal function is to stimulate the metabolism of all cells and tissues in the body. Excessive secretion of thyroxine in the body is hyperthyroidism. And deficient exception is called by prototypical fraction of the thyroid hormone in blood is hard to transport proteins. Only a yeary small fraction of the

hyperthyroidism, and deficient secretion is called 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.

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







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Levels in TOTAL T4 TSH3G TOTAL T3 (μIU/mL) 0.1 - 2.5 0.2 - 3.0 0.3 - 3.0 (µg/dL) 6.6 - 12.4 (ng/dL) 81 - 190 Pregnancy First Trimester 100 - 260 100 - 260 2nd Trimester 6.6 - 15.53rc Trimester 6.6 - 15.5 Below mentioned are the guidelines for age related reference ranges for T3 and T4.

ТЗ (ng/dL) (µg/dL)

New Born: 75 - 260 1-3 day: 8.2 - 19.9 1 Week: 6.0 - 15.9

NOTE: TSH concentrations in apparently normal euthyroid subjects are known to be highly skewed, with a strong tailed distribution towards higher TSH values. This is well documented in the pediatric population including the infant age group.

Kindly note: Method specific reference ranges are appearing or the report under biological reference range.

- Reference:

 1. Burtis C.A., Ashwood E.R. Bruns D.E. Teitz textbook of Clinical Chemistry and Molecular Diagnostics, 4th Edition.

 2. Gowenlock A.H. Varley's Practical Clinical Biochemistry, 6th Edition.

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 ABO GROUP & RH TYPE, EDTA WHOLE BLOOD-

Blood group is identified by artigens 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: "Flease note, as the results of previous ABO and Rh group (Blood Group) for pregnant women are not available, please check with the patient records for availability of the same

The test is performed by both forward as well as reverse grouping methods.

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







CLIENT'S NAME AND ADDRESS:

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MEDI WHEEL FULL BODY HEALTH CHECK UP ABOVE 40 MALE

ULTRASOUND ABDOMEN ULTRASOUND ABDOMEN GRADE | FATTY LIVER. LEFT RENAL CALCULUS. MILD PROSTATOMEGALY.

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

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