

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: MUKESHK. DIWAKAR

PATIENT ID: MUKEM15057240

ACCESSION NO: 0181VI000302 AGE: 50 Years SEX: Male

DRAWN: RECEIVED: 10/09/2022 09:13 REPORTED: 15/09/2022 15:34

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 | 1.005 | | 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. /1 | | 4.5 - 5.5 | mil/µL |
| METHOD: HYDRODYNAMIC FOCUSING BY DC DETECTION | | | | |
| WHILE BLOOD CELL COUNT | 4.14 | | 4.0 - 10.0 | thou/µL |
| METHOD: FLUORESCENCE FLOW CYTOMETRY | | | | |
| PLATELET COUNT | 133 | Low | 150 - 410 | thou/µL |
| METHOD: HYDRODYNAMIC FOCUSING BY DC DETECTION | | | | |
| RBC AND PLATELET INDICES | | | | |
| HEMATOCRIT | 44.0 | | 40.0 - 50.0 | % |
| METHOD : CUMULATIVE PULSE HEIGHT DETECTION METHOD | | | | |
| MEAN CORPUSCULAR VOL | 93.4 | | 83.0 - 101.0 | tL |
| METHOD : CALCULATED FROM RBC & HCT | | | | |
| MEAN CORPUSCULAR HGB. | 30.1 | | 27.0 - 32.0 | pg |
| METHOD: CALCULATED FROM THE RBC & HGB | | | | |
| MEAN CORPUSCULAR HEMOGLOBIN | 32.3 | | 31.5 - 34.5 | g/dL |
| CONCENTRATION METHOD: CALCULATED FROM THE HGB & HCT | | | | |
| MENTZER INDEX | 19.8 | | | |
| RED CELL DISTRIBUTION WIDTH | 14.3 | Hiah | 11.6 - 14.0 | % |
| METHOD : CALCULATED FROM RBC SIZE DISTRIBUTION CURVE | 1110 | | 1110 1110 | ,0 |
| MEAN PLATELET VOLUME | 14.8 | High | 6.8 - 10.9 | fL |
| METHOD : CALCULATED FROM PLATELET COUNT & PLATELET HEMA | | _ | | |
| CHEMICAL EXAMINATION, URINE | | | | |
| P⊢ | 7.0 | | 4.7 - 7.5 | |
| METHOD : DOUBLE INDICATOR PRINCIPLE | · · - | | | |
| PROTEIN | NOT DETECTED | | 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 | NORMA | | 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 | ć. 4 | | 40 00 | 0.4 |
| SEGMENTED NEUTROPHILS | 64 | | 40 - 80 | % |
| METHOD: FLOW CYTOMETRY WITH LIGHT SCATTERING | 2.65 | | 20.70 | He ex color |
| ABSOLUTE NEUTROPHIL COUNT | 2.65 | | 2.0 - 7.0 | thou/µL |
| METHOD: FLOW CYTOMETRY WITH LIGHT SCATTERING LYMPHOCYTES | 30 | | 20 - 40 | % |
| METHOD: FLOW CYTOMETRY WITH LIGHT SCATTERING | 50 | | 20 - 40 | 70 |
| ABSOLUTE LYMPHOCYTE COUNT | 1.24 | | 1.0 - 3.0 | thou/µL |
| METHOD: FLOW CYTOMETRY WITH LIGHT SCATTERING | 1.27 | | 1.0 3.0 | a 100, pc |
| NEUTROPHIL LYMPHOCYTE RATIC (NLR) | 2.1 | | | |
| EOSINOPHILS | 03 | | 1 - 6 | % |
| METHOD: FLOW CYTOMETRY WITH LIGHT SCATTERING | 00 | | 1 0 | ,0 |
| ABSOLUTE EOSINOPHIL COUNT | 0.12 | | 0.02 - 0.50 | thou/µL |
| METHOD: FLOW CYTOMETRY WITH LIGHT SCATTERING | | | | |
| MONOCYTES | 03 | | 2 - 10 | % |
| METHOD: FLOW CYTOMETRY WITH LIGHT SCATTERING | | | | |
| ABSOLUTE MONOCYTE COUNT | 0.12 | Low | 0.2 - 1.0 | thou/µL |
| METHOD: FLOW CYTOMETRY WITH LIGHT SCATTERING | | | | |
| DIFFERENTIAL COUNT PERFORMED ON: | EDTA SMEAR | | | |
| MICROSCOPIC EXAMINATION, URINE | | | | |
| PUS CELL (WBC'S) | 1-2 | | 0-5 | /HPF |
| METHOD: MICROSCOPIC EXAMINATION | | | | |
| EPITHELIAL CELLS | 1-2 | | 0-5 | /HPF |
| METHOD: MICROSCOPIC EXAMINATION | | | | |
| ERYTHROCYTES (RBC'S) | NOT DETECTED | | NOT DETECTED | /HPF |
| METHOD: MICROSCOPIC EXAMINATION | | | | |
| CASTS | NOT DETECTED | | | |







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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 | |
| MORPHOLOGY | | | |
| RBC | PREDOMINANTLY NORMOCYTIC NORMOCHROMIC | | |
| WBC | NORMAL MORPHOLOGY | | |
| METHOD: MICROSCOPIC EXAMINATION | | | |
| PLATELETS | REDUCED, PLATELET COUNT ON SMEAR APPEARS TO BE 130-140 THOU/UL, GIANT PLATELETS SEEN | | |
| ERYTHRO SEDIMENTATION RATE, BLOOD | | | |
| SEDIMENTATION RATE (ESR) | 02 | 0 - 14 | mm at 1 hr |
| METHOD: WESTERGREN METHOD | | | |
| GLYCOSYLATED HEMOGLOBIN, EDTA WHOLE BL | _OOD | | |
| GLYCOSYLATED HEMOGLOBIN (HBA1C) | 5.0 | Non-diabetic: < 5.7 Pre-diabetics: 5.7 - 6.4 Diabetics: > or = 6.5 ADA Target: 7.0 Action suggested: > 8.0 | % |
| METHOD: HPLC | | | |
| MEAN PLASMA GLUCOSE | 96.8 | < 116.0 | mg/dL |
| METHOD: CALCULATED PARAMETER | | | |
| GLUCOSE, FASTING, PLASMA | | | |
| GLUCOSE, FASTING, PLASMA | 87 | Normal 75 - 99 Pre-diabetics: 100 - 125 Diabetic: > or = 126 | mg/dL |
| METHOD: ENZYMATIC REFERENCE METHOD WITH HEXOKINASE | | | |
| GLUCOSE, POST-PRANDIAL, PLASMA | | | |
| GLUCOSE, POST-PRANDIAL, PLASMA | 113 | 70 - 139 | mg/dL |
| METHOD: ENZYMATIC REFERENCE METHOD WITH HEXOKINASE | | | |
| CORONARY RISK PROFILE, SERUM | | | |
| CHOLESTEROL | 209 High | Desirable cholesterol level < 200 Borderline high cholesterol 200 - 239 | mg/dL |

METHOD: ENZYMATIC COLORIMETRIC ASSAY



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High cholesterol > / = 240



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| TRIGLYCERIDES METHOD: ENZYMATIC COLORIMETRIC ASSAY | 97 | | Normal: < 150 Borderline high: 150 - 199 High: 200 - 499 Very High: >/= 500 | mg/dL |
| HDL CHOLESTEROL | 48.6 | | Low HDL Cholesterol <40 | mg/dL |
| METHOD : ENZYMATIC, COLORIMETRIC | 1010 | | High HDL Cholesterol >/= 60 | - |
| CHOLESTEROL LDL | 141 | High | Adult levels: Optimal < 100 Near optimal/above optimal: 1 129 Borderline high: 130-159 High: 160-189 Very high: = 190 | mg/dL 00- |
| METHOD: ENZYMATIC COLORIMETRIC ASSAY | | | . 0 | |
| NON HDL CHOLESTEROL | 160 | High | Desirable: < 130 Above Desirable: 130 -159 Borderline High: 160 - 189 High: 190 - 219 Very high: > / = 220 | mg/dL |
| CHOL/HDL RATIO LDL/HDL RATIO | 4.3 2.9 | | Low Risk: 3.3 - 4.4 Average Risk: 4.5 - 7.0 Moderate Risk: 7.1 - 11.0 High Risk: > 11.0 0.5 - 3.0 Desirable/Low Risk | Diele |
| | | | 3.1 - 6.0 Borderline/Moderate > 6.0 High Risk | KISK |
| VERY LOW DENSITY LIPOPROTEIN | 19.4 | | < OR = 30.0 | mg/dL |
| LIVER FUNCTION PROFILE, SERUM | | | | |
| BILIRUBIN, TOTAL METHOD: COLORIMETRIC DIAZO | 0.81 | | Upto 1.2 | mg/dL |
| BILIRUBIN, DIRECT | 0.30 | | < 0.30 | mg/dL |
| BILIRUBIN, INDIRECT | 0.51 | | 0.1 - 1.0 | mg/dL |
| TOTAL PROTEIN METHOD: COLORIMETRIC | 7.7 | | 6.0 - 8.0 | g/dL |
| ALBUMIN METHOD: COLORIMETRIC | 5.1 | High | 3.97 - 4.94 | g/dL |
| GLOBULIN | 2.6 | | 2.0 - 3.5 | g/dL |
| ALBUMIN/GLOBULIN RATIO | 2.0 | | 1.0 - 2.1 | RATIO |
| ASPARTATE AMINOTRANSFERASE (AST/SGOT) METHOD: UV ABSORBANCE | 26 | | < OR = 50 | U/L |







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| ALANINE AMINOTRANGEEDAGE (ALT/CODT) | 25 | | 4.0D F0 | 112 |
| ALANINE AMINOTRANSFERASE (ALT/SGPT) METHOD: UV ABSORBANCE | 26 | | < OR = 50 | U/L |
| ALKALINE PHOSPHATASE | 102 | | 40 - 129 | U/L |
| METHOD : COLORIMETRIC | 102 | | 123 | 0, L |
| GAMMA GLUTAMYL TRANSFERASE (GGT) | 15 | | 0 - 60 | U/L |
| METHOD : ENZYMATIC, COLORIMETRIC | | | | |
| LACTATE DEHYDROGENASE | 188 | | 125 - 220 | U/L |
| METHOD: UV ABSORBANCE | | | | |
| SERUM BLOOD UREA NITROGEN | | | | |
| BLOOD UREA NITROGEN | 7 | | 6 - 20 | mg/dL |
| METHOD: ENZYMATIC ASSAY | | | | |
| CREATININE, SERUM | | | | |
| CREATININE | 1.01 | | 0.7 - 1.2 | mg/dL |
| METHOD : COLORIMETRIC | | | | |
| BUN/CREAT RATIO | | | | |
| BUN/CREAT RATIO | 6.93 | Low | 8.0 - 15.0 | |
| URIC ACID, SERUM | | | | |
| URIC ACID | 6.9 | | 3.4 - 7.0 | mg/dL |
| METHOD : ENZYMATIC COLORIMETRIC ASSAY | | | | |
| TOTAL PROTEIN, SERUM | | | | |
| TOTAL PROTEIN | 7.7 | | 6.0 - 8.0 | g/dL |
| METHOD : COLORIMETRIC | | | | |
| ALBUMIN, SERUM | F 1 | | 0.07 4.04 | f. II |
| ALBUMIN | 5.1 | High | 3.97 - 4.94 | g/dL |
| METHOD : COLORIMETRIC GLOBULIN | | | | |
| GLOBULIN | 2.6 | | 2.0 - 3.5 | a/dL |
| | ∠.0 | | 2.0 - 3.3 | g/uL |
| ELECTROLYTES (NA/K/CL), SERUM | 1.44 | | 106 145 | marea a l /l |
| SODIUM | 144 | | 136 - 145 | mmol/L |
| POTASSIUM | 4.66 | | 3.5 - 5.1 | mmol/L |
| CHLORIDE | 105 | | 98 - 107 | mmol/L |
| THYROID PANEL, SERUM | | | | |
| T3 | 62.3 | Low | 80 - 200 | ng/dL |
| METHOD : ELECTROCHEMILUMINESCENCE | 4.67 | Law | 51 141 | الحارض |
| T4 | 4.67 | LOW | 5.1 - 14.1 | μg/dL |
| METHOD : ELECTROCHEMILUMINESCENCE TSH 3RD GENERATION | 2.750 | | 0.27 - 4.2 | μΙU/mL |
| TOTTONE GENERALION | 2.750 | | 0,27 - 4,2 | h10/IIIC |







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METHOD: ELECTROCHEMILUMINESCENCE

STOOL: OVA & PARASITE

COLOUR **BROWN**

METHOD: VISUAL

CONSISTENCY WELL FORMED

METHOD: VISUAL

ODOUR FAECAL

METHOD: PHYSICAL

MUCUS ABSENT NOT DETECTED

METHOD: VISUAL

VISIBLE BLOOD ABSENT **ABSENT**

METHOD: VISUAL

POLYMORPHONUCLEAR LEUKOCYTES 0 - 10 - 5 /HPF

METHOD: MICROSCOPIC EXAMINATION

RED BLOOD CELLS NOT DETECTED NOT DETECTED /HPF

METHOD: MICROSCOPIC EXAMINATION

TROPHOZOITES NOT DETECTED NOT DETECTED

METHOD: MICROSCOPIC EXAMINATION

CYSTS NOT DETECTED NOT DETECTED

METHOD: MICROSCOPIC EXAMINATION

METHOD: MICROSCOPIC EXAMINATION

LARVAE NOT DETECTED NOT DETECTED

METHOD: MICROSCOPIC EXAMINATION

OCCULT BLOOD NOT DETECTED NOT DETECTED

METHOD: HEMOSPOT

REMARK NO OVA CYST SEEN AFTER PERFORMING CONCENTRATION TECHNIQUE

NOT DETECTED

FOR STOOL SAMPLE

ABO GROUP & RH TYPE, EDTA WHOLE BLOOD

ABO GROUP TYPE B

METHOD: GEL COLUMN AGGLUTINATION METHOD.

RH TYPE **POSITIVE**

METHOD: GEL COLUMN AGGLUTINATION METHOD.

XRAY-CHEST

IMPRESSION NO ABNORMALITY DETECTED

TMT OR ECHO

TMT OR ECHO NEGATIVE

ECG



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SEX: Male

ECG LEFT AXIS DEVIATION.

MEDICAL HISTORY

RELEVANT PRESENT HISTORY NOT SIGNIFICANT

OPERATED FOR PILES IN 2016. RELEVANT PAST HISTORY

PAST H/O JAUNDICE TWICE

COVID TWICE IN THE PAST. HOME QUARANTINED.

RELEVANT PERSONAL HISTORY MARRIED / 2 CHILD / VEG DIET / NO ALLERGIES / NO SMOKING / NC

ALCOHOL.

RELEVANT FAMILY HISTORY HIGH BLOOD PRESURE: MOTHER.

HEART DISEASE: FATHER.

HISTORY OF MEDICATIONS NOT SIGNIFICANT

ANTHROPOMETRIC DATA & BMI

HEIGHT IN METERS 1.70 mts WEIGHT IN KGS. 76 Kas

RMI BMI & Weight Status as follows: kg/samts 26

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** NORMAL FACIAL APPEARANCE SKIN NORMAL UPPER LIMB NORMAL **LOWER ITMB** NORMAL NECK NORMAL

NECK LYMPHATICS / SALIVARY GLANDS NOT ENLARGED OR TENDER

THYROID GLAND NOT ENLARGED

CAROTID PULSATION NORMAL TEMPERATURE NORMAL

PULSE 68/MIN.REGULAR, ALL PERIPHERAL PULSES WELL FELT, NO CAROTID

BRUIT

RESPIRATORY RATE NORMAL

CARDIOVASCULAR SYSTEM







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| BP | 114/80 MM HG (SUPINE) | mr | m/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 | | |
| DISTANT VISION RIGHT EYE WITHOUT GLASSES | REDUCED VISUAL ACUITY | 5/9 | |

REDUCED VISUAL ACUITY 6/9



DISTANT VISION LEFT EYE WITHOUT GLASSES





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NEAR VISION RIGHT EYE WITHOUT GLASSES REDUCED VISUAL ACUITY N/36 NEAR VISION LEFT EYE WITHOUT GLASSES REDUCED VISUAL ACUITY N/36

NEAR VISION RIGHT EYE WITH GLASSES WITHIN NORMAL LIMIT NEAR VISION LEFT EYE WITH GLASSES WITHIN NORMAL LIMIT

COLOUR VISION NORMAL

SUMMARY

RELEVANT HISTORY NOT SIGNIFICANT RELEVANT GP EXAMINATION FINDINGS NOT SIGNIFICANT

REMARKS / RECOMMENDATIONS ADVICE: -

1) LOW FAT, LOW CALORIE, LOW CARBOHYDRATE, HIGH FIBRE DIET,

2) REGULAR EXERCISE.REGULAR WALK FOR 30-40 MIN DAILY.

REPEAT LIPID, THYROID PROFILE PROFILE AFTER 3 MONTHS OF DIET

AND EXERCISE.

4) REPEAT CBC AFTER 1 MONTH.

5)TO DO S.PSA.

6) UROLOGIST CONSULT IN VIEW OF PROSTATOMEGALY.

Interpretation(s)
BLOOD COUNTS,EDTA WHOLE BLOOD-

The cell morphology is well preservec 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 - NLR-

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

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

Protein: Elevated proteins car be an early sign of kidney disease. Urinary protein excretion can also be temporarily elevated by strenuous exercise, orthostatic proteinuria, dehydration, urmary 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

Ketones: Uncontrolled diabetes mellitus can 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 infection. 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 acic 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 concentratec the urine is. Increasec specific gravity is seen in conditions like dehydration, glycosuria and proteinum 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 unine.

Urobilinogen: Positive results are seen in liver diseases like hepatitis and cirrhosis and in cases of hemolytic anemia ERYTHRO SEDIMENTATION RATE, BLOOD-

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, hypothorinogenemia or congestive cardiac failure and when there are abnormalities of the red cells such as polikilocytosis, spherocytosis or sickle cells.







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PATIENT NAME: MUKESH K. DIWAKAR

PATIENT ID: MUKEM15057240

ACCESSION NO: 0181VI000302 AGE: 50 Years SEX: Male

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

Reference:

1. Nathan and Oski's Haematology of Infancy and Childhood, 5th edition 2. Paediatric reference intervals. AACC Press, 7th edition. Edited by S. Soldin

3. The reference for the adult reference range is "Practical Haematology by Dacie and Lewis, 10th Edition" GLYCOSYLATED HEMOGLOBIN, 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,

blood 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 afters 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 hemoglobin 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 increased glycated hemoglobin values due to a somewhat longer life span of the red cells.

Glycosylatec hemoglobins results from patients with HbSS, HbCC, and HbSC and HbD must be interpreted with caution, given the pathological processes, including anemia, increased red 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 (tructosamine) 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

1. Tietz Textbook of Clinical Chemistry and Molecular Diagnostics, edited by Carl A Burbs, Edward R.Ashwood, David E Bruns, 4th Edition, Elsevier publication, 2006, 879-884.

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

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 minutes.

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 bilirubir production (eg, hemolysis and ineffective erythropolesis), 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 dysfunction. Elevated serum GGT activity can be found in diseases of the liver, billiary system and pandreas. Conditions that increase serum GGT are obstructive liver disease, high alcohol consumption 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 albumin 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, Glomerulonephritis, Liver disease, Malabsorption, Malnutrition, Nephrotic syndrome, Protein-losing enteropathy etc. 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

SERUM BLOOD UREA NITRÓGEN-

Causes of Increased levels

Pre renal

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

· Malignancy, Nephrolithiasis, Prostatism

Causes of decreased levels

Liver disease



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• 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 DM. Metabolic syndrome.

Causes of decreased levels

- Low Zinc Intake
- OCP's
- Multiple Scierosis

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

- Lmit 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-losing enteropathy etc. ALBUMIN, SERUM-

Human serum albumir is the most abundant protein in human blooc plasma. It is produced in the liver. Albumin constitutes about half of the blooc 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, mainutrition and wasting etc. ELECTROLYTES (NA/K/CL), SERUM-

Sodium 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 respiratory acidosis, salt-losing nephritis, metabolic alkalosis, congestive heart failure, Addisoniar crisis, certain types of metabolic acidosis, persistent gastric secretion and

prolongec vomiting, THYROID PANEL, SERUM-

Trillodothyronine 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 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

Levels in TOTAL T4 ŤSHЗĠ TOTAL T3 (µg/dL) 6.6 - 12.4 6.6 - 15.5 (ng/dL) 81 - 190 100 - 260 Pregnancy (µIU/mL) 0.1 - 2.5 First Trimester 2nd Trimester 3rc Trimester 6.6 - 15.50.3 - 3.0100 - 260



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Below mentioned are the guidelines for age related reference ranges for T3 and T4.

Т3 Τ4 (µg/dL) 1-3 day: 8.2 - 19.9 1 Week: 6.0 - 15.9 (ng/dL) New Born: 75 - 260

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 on the report under biological reference range.

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

Burns C.A., Astroyou F. R. Brus B.E. Felz exhabits of crimeal crimeal of a library and molecular 2. Gowenlock A.H. Varley's Pactical Clinical Biochemistry, 6th Edition.
 Behrman R.E. Kilegman R.M., Jenson H. B. Nelson Text Book of Pediatrics, 17th Edition.

STOOL: OVA & PARASITE-

Acute infective diarrhoea and gastroenterits (diarrhoea with vomiting) are major causes of ill health and premature death in developing countries. Loss of water and electrolytes from the body can lead to severe dehydration which it untreated, can be rapidly fatal in young children, especially that are malnourished, hypoglycaemic, and generally in poor health

Laboratory diagnosis of parasitic infection is mainly based on microscopic examination and the gross examination of the stool specimen. Depending on the nature of the parasite, the microscopic observations include the identification of cysts, ova, trophozoites, larvae or portions of adult structure. The two classes of parasites that cause human infection are the Protozoa and Helminths. The protozoan infections include amoeblasis mainly caused by Entamoeba histolytica and giardiasis caused by Giardia lamblia. The common helminthic parasites are Trichuris trichiura, Ascaris lumbricoides, Strongyloides stercoralis, Taenia sp. etc
ABO GROUP & RH TYPE, EDTA WHOLE BLOODBlooc 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: "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.







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MEDI WHEEL FULL BODY HEALTH CHECK UP ABOVE 40 MALE

ULTRASOUND ABDOMEN

ULTRASOUND ABDOMEN

GRADE | FATTY LIVER.

MODERATE PROSTATOMEGALY WITH MILD MEDIAN LOBE HYPERTROPHY.

SIGNIFICANT POST VOID RESIDUE.

ADVICE: - S.PSA CORRELATION.

End Of Report

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

Dr. Sheetal Sawant Consultant Microbiologist

Dr. Ushma Wartikar Consultant Pathologist Dr.(Mrs)Neelu K Bhojani

Lab Head



