



CLIENT CODE: C000138383 **CLIENT'S NAME AND ADDRESS:**

ACROFEMI HEALTHCARE LTD (MEDIWHEEL)

F-703, LADO SARAI, MEHRAULI SOUTH WEST DELHI **NEW DELHI 110030**

DELHI INDIA 8800465156 SRL Ltd

24 SCO, SECTOR 11 D CHANDIGARH, 160011 PUNJAB, INDIA Tel: 9111591115, Fax:

CIN - U74899PB1995PLC045956

PATIENT NAME: SUNIL KUMAR 528408607650 PATIENT ID: SUNIM11068580

ACCESSION NO: **0080VG007192** AGE: 37 Years SEX: Male ABHA NO:

RECEIVED: 16/07/2022 09:43 16/07/2022 16:54 DRAWN: REPORTED:

REFERRING DOCTOR: SELF CLIENT PATIENT ID:

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

MEDI WHEEL FULL BODY HEALTH CHECK UP BELOW 40 MALE

BLOOD COUNTS,EDTA WHOLE BLOOD				
HEMOGLOBIN	14.5		13.0 - 17.0	g/dL
RED BLOOD CELL COUNT	5.17		4.5 - 5.5	mil/μL
WHITE BLOOD CELL COUNT	4.10		4.0 - 10.0	thou/µL
PLATELET COUNT	191		150 - 410	thou/µL
RBC AND PLATELET INDICES				
HEMATOCRIT	43.2		40.0 - 50.0	%
METHOD: ELECTRICAL IMPEDANCE				
MEAN CORPUSCULAR VOL	83.6		83.0 - 101.0	fL
MEAN CORPUSCULAR HGB.	28.1		27.0 - 32.0	pg
MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION	33.6		31.5 - 34.5	g/dL
MENTZER INDEX	16.2			
RED CELL DISTRIBUTION WIDTH	13.6		11.6 - 14.0	%
MEAN PLATELET VOLUME	9.6		6.8 - 10.9	fL
WBC DIFFERENTIAL COUNT - NLR				
SEGMENTED NEUTROPHILS	45		40 - 80	%
ABSOLUTE NEUTROPHIL COUNT	1.85	Low	2.0 - 7.0	thou/µL
LYMPHOCYTES	42	High	20 - 40	%
ABSOLUTE LYMPHOCYTE COUNT	1.72		1.0 - 3.0	thou/µL
NEUTROPHIL LYMPHOCYTE RATIO (NLR)	1.1			
METHOD: CALCULATED PARAMETER				
EOSINOPHILS	5		1 - 6	%
ABSOLUTE EOSINOPHIL COUNT	0.21		0.02 - 0.50	thou/µL
MONOCYTES	7		2 - 10	%
ABSOLUTE MONOCYTE COUNT	0.29		0.2 - 1.0	thou/µL
METHOD: CALCULATED PARAMETER				
BASOPHILS	1		0 - 1	%
ABSOLUTE BASOPHIL COUNT	0.04		0.02 - 0.10	thou/µL
METHOD: CALCULATED PARAMETER				
DIFFERENTIAL COUNT PERFORMED ON:	AUTOMATED ANALYZ	ER.		
ERYTHRO SEDIMENTATION RATE, BLOOD				
SEDIMENTATION RATE (ESR)	05		0 - 14	mm at 1 hr
METHOD: MODIFIED WESTERGREN				

METHOD: MODIFIED WESTERGREN





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CLUCOCE FACTING DIAGNA				
GLUCOSE, FASTING, PLASMA	0.5		74 405	
GLUCOSE, FASTING, PLASMA	95		74 - 106	mg/dL
METHOD: HEXOKINASE				
GLYCOSYLATED HEMOGLOBIN, EDTA WHO	DLE BLOOD			
GLYCOSYLATED HEMOGLOBIN (HBA1C)	5.4		Non-diabetic: < 5.7 Pre-diabetics: 5.7 - 6.4 Diabetics: > or = 6.5 ADA Target: 7.0 Action suggested: > 8.0	%
MEAN PLASMA GLUCOSE	108.3		< 116.0	mg/dL
GLUCOSE, POST-PRANDIAL, PLASMA				
GLUCOSE, POST-PRANDIAL, PLASMA	91		Non-Diabetes 70 - 140	mg/dL
METHOD: HEXOKINASE				
CORONARY RISK PROFILE (LIPID PROFIL	E), SERUM			
CHOLESTEROL	218	High	< 200 Desirable 200 - 239 Borderline High >/= 240 High	mg/dL
METHOD: CHOLESTEROL OXIDASE, ESTERASE, PEROXIDAS	SE .		,	
TRIGLYCERIDES	187	High	< 150 Normal 150 - 199 Borderline High 200 - 499 High >/= 500 Very High	mg/dL
METHOD: ENZYMATIC ASSAY				
HDL CHOLESTEROL	50		< 40 Low >/=60 High	mg/dL
METHOD : DIRECT MEASURE - PEG			2/ =00 High	
DIRECT LDL CHOLESTEROL METHOD: CHOLESTEROL OXIDASE, ESTERASE, PEROXIDAS	165	High	< 100 Optimal 100 - 129 Near or above option 130 - 160 Borderline High 161 - 189 High >/= 190 Very High	mg/dL mal
NON HDL CHOLESTEROL	168	High	Desirable: Less than 130	mg/dL
	105	911	Above Desirable: 130 - 159 Borderline High: 160 - 189 High: 190 - 219 Very high: > or = 220	mg/uL
METHOD : CALCULATED PARAMETER			22441	
CHOL/HDL RATIO	4.4		3.3-4.4 Low Risk 4.5-7.0 Average Risk 7.1-11.0 Moderate Risk > 11.0 High Risk	
METHOD: CALCULATED PARAMETER				

METHOD: CALCULATED PARAMETER









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LDL/HDL RATIO	3.3	High	0.5 - 3.0 Desirable/Low Risk 3.1 - 6.0 Borderline/Moderate Risk >6.0 High Risk	
METHOD : CALCULATED PARAMETER VERY LOW DENSITY LIPOPROTEIN	37.4	High	Desirable value : 10 - 35	mg/dL
METHOD : CALCULATED PARAMETER			10 - 35	
LIVER FUNCTION PROFILE, SERUM				
BILIRUBIN, TOTAL	0.94		UPTO 1.2	mg/dL
METHOD : DIAZONIUM ION, BLANKED (ROCHE)				
BILIRUBIN, DIRECT	0.23		0.00 - 0.30	mg/dL
METHOD : DIAZOTIZATION				
BILIRUBIN, INDIRECT	0.71	High	0.00 - 0.60	mg/dL
METHOD: CALCULATED PARAMETER				
TOTAL PROTEIN	7.4		6.6 - 8.7	g/dL
METHOD : BIURET				
ALBUMIN	4.7		3.97 - 4.94	g/dL
METHOD: BROMOCRESOL GREEN				
GLOBULIN	2.7		2.0 - 4.0 Neonates - Pre Mature: 0.29 - 1.04	g/dL
METHOD: CALCULATED PARAMETER				
ALBUMIN/GLOBULIN RATIO	1.7		1.0 - 2.0	RATIO
METHOD : CALCULATED PARAMETER				
ASPARTATE AMINOTRANSFERASE (AST/SGOT)	22		0 - 40	U/L
ALANINE AMINOTRANSFERASE (ALT/SGPT)	26		0 - 41	U/L
METHOD: UV WITHOUT PYRIDOXAL-5 PHOSPHATE				
ALKALINE PHOSPHATASE	75		40 - 129	U/L
METHOD: PNPP - AMP BUFFER	4.6		0 61	11.7
GAMMA GLUTAMYL TRANSFERASE (GGT)	46		8 - 61	U/L
METHOD: GAMMA GLUTAMYLCARBOXY 4NITROANILIDE LACTATE DEHYDROGENASE	193		135 - 225	U/L
METHOD : LACTATE -PYRUVATE	193		133 - 223	U/L
SERUM BLOOD UREA NITROGEN				
BLOOD UREA NITROGEN	10		6 - 20	mg/dL
METHOD : UREASE - UV	10		0 20	ilig/uL
CREATININE, SERUM				
CREATININE CREATININE	0.89		0.70 - 1.20	mg/dL
METHOD : ALKALINE PICRATE-KINETIC	0.03		0.70 1.20	ilig/uL
MEIGRETTE LIGITURE MINERIO				



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BUN/CREAT RATIO				
BUN/CREAT RATIO		11.24	5.00 - 15.00	
METHOD : CALCULATED PAR	ΔMETER	11.27	5.00 15.00	
URIC ACID, SERUM				
URIC ACID		5.7	3.4 - 7.0	mg/dL
METHOD : URICASE, COLOR	IMETRIC	3.,	311 710	1119, 42
TOTAL PROTEIN, SER				
TOTAL PROTEIN		7.4	6.6 - 8.7	g/dL
METHOD : BIURET				37
ALBUMIN, SERUM				
ALBUMIN		4.7	3.97 - 4.94	g/dL
METHOD : BROMOCRESOL G	REEN			3,
GLOBULIN				
GLOBULIN		2.7	2.0 - 4.0 Neonates - Pre Mature: 0.29 - 1.04	g/dL
METHOD : CALCULATED PAR	AMETER		0.25 1.01	
ELECTROLYTES (NA/	K/CL), SERUM			
SODIUM		137	136 - 145	mmol/L
METHOD : ISE INDIRECT				
POTASSIUM		4.32	3.5 - 5.1	mmol/L
METHOD : ISE INDIRECT				
CHLORIDE		100	98 - 107	mmol/L
METHOD: ISE INDIRECT				
PHYSICAL EXAMINAT	ΓΙΟΝ, URINE			
COLOR		PALE YELLOW		
APPEARANCE		CLEAR		
SPECIFIC GRAVITY		1.030	1.003 - 1.035	
METHOD : REFLECTANCE SP	ECTROPHOTOMETRY (PKA CHAN	IGE OF PRETREATED POLY ELECTROLYTES)		
CHEMICAL EXAMINA	TION, URINE			
PH		6.0	4.7 - 7.5	
METHOD : REFLECTANCE SPI	ECTROPHOTOMETRY- DOUBLE I	NDICATOR METHOD		
PROTEIN		NOT DETECTED	NOT DETECTED	
METHOD: REFLECTANCE SPI	ECTROPHOTOMETRY (PROTEIN-	ERROR-OF-INDICATORS PRINCIPLE)		
GLUCOSE		NOT DETECTED	NOT DETECTED	
	ECTROPHOTOMETRY(GLUCOSE	OXIDAE/PEROXIDASE METHOD)		
KETONES		NOT DETECTED	NOT DETECTED	









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METHOD - REFLECTANCE S	PECTROPHOTOMETRY (SOF	DIUM NITROPRUSSIDE REACTION)		
BLOOD	FLCTROFFIOTOMETRI (30L	NOT DETECTED	NOT DETECTED	
METHOD : REFLECTANCE S	PECTROPHOTOMETRY (PER		NOT BETEGIED	
BILIRUBIN		NOT DETECTED	NOT DETECTED	
METHOD : REFLECTANCE S	PECTROPHOTOMETRY (DIA			
UROBILINOGEN	`	NORMAL	NORMAL	
METHOD : REFLECTANCE S	PECTROPHOTOMETRY - EHI			
NITRITE		NOT DETECTED	NOT DETECTED	
METHOD : REFLECTANCE S	PECTROPHOTOMETRY, CON	IVERSION OF NITRATE TO NITRITE		
LEUKOCYTE ESTERASI		NOT DETECTED	NOT DETECTED	
MICROSCOPIC EXAM	INATION, URINE			
PUS CELL (WBC'S)	·	0-1	0-5	/HPF
METHOD : MICROSCOPIC E	XAMINATION			,
EPITHELIAL CELLS		0-1	0-5	/HPF
METHOD : MICROSCOPIC E	XAMINATION			·
ERYTHROCYTES (RBC'	S)	NOT DETECTED	NOT DETECTED	/HPF
METHOD : MICROSCOPIC E	XAMINATION			
CASTS		NOT DETECTED		
CRYSTALS		NOT DETECTED		
METHOD : MICROSCOPIC E	XAMINATION			
BACTERIA		NOT DETECTED	NOT DETECTED	
METHOD : MICROSCOPIC E	XAMINATION			
YEAST		NOT DETECTED	NOT DETECTED	
THYROID PANEL, SE	RUM			
T3		96.6	80.00 - 200.00	ng/dL
METHOD : COMPETITIVE (E	CLIA)			-
T4		6.26	5.10 - 14.10	μg/dL
METHOD : COMPETITIVE (E	CLIA)			
TSH 3RD GENERATION	J	1.610	0.270 - 4.200	μIU/mL
METHOD : SANDWICH (ECL	IA)			
ABO GROUP & RH T	PE, EDTA WHOLE	BLOOD		
ABO GROUP		TYPE B		
METHOD : TUBE AGGLUTIN	ATION			
RH TYPE		POSITIVE		
METHOD : TUBE AGGLUTIN	ATION			



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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 - 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.

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

- 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" 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

GLYCOSYLATED HEMOGLOBIN, EDTA WHOLE BLOOD-

Glycosylated 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 red 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 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 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.

Glycosylated 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 (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, 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.

COROMARY RISK PROFILE (LIPID PROFILE), SERUM-Serum cholesterol is a blood test that can provide valuable information for the risk of coronary artery disease This test can help determine your risk of the build up of plaques in your arteries that can lead to narrowed or blocked arteries throughout your body (atherosclerosis). High cholesterol levels usually don't cause any signs or symptoms, so a cholesterol test is an important tool. High cholesterol levels often are a significant risk factor for heart disease and important for diagnosis of hyperlipoproteinemia, atherosclerosis, hepatic and thyroid diseases.

Serum Triglyceride are a type of fat in the blood. When you eat, your body converts any calories it doesn"t need into triglycerides, which are stored in fat cells. High triglyceride levels are associated with several factors, including being overweight, eating too many sweets or drinking too much alcohol, smoking, being sedentary, or having diabetes with elevated blood sugar levels. Analysis has proven useful in the diagnosis and treatment of patients with diabetes mellitus, nephrosis, liver obstruction, other diseases involving lipid metabolism, and various endocrine disorders. In conjunction with high density lipoprotein and total serum cholesterol, a triglyceride determination provides valuable information for the assessment of coronary heart disease risk. It is done in fasting state.

High-density lipoprotein (HDL) cholesterol. This is sometimes called the ""good"" cholesterol because it helps carry away LDL cholesterol, thus keeping arteries open and blood flowing more freely.HDL cholesterol is inversely related to the risk for cardiovascular disease. It increases following regular exercise, moderate alcohol consumption and with oral estrogen therapy. Decreased levels are associated with obesity, stress, cigarette smoking and diabetes mellitus.

SERUM LDL The small dense LDL test can be used to determine cardiovascular risk in individuals with metabolic syndrome or established/progressing coronary artery



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disease, individuals with triglyceride levels between 70 and 140 mg/dL, as well as individuals with a diet high in trans-fat or carbohydrates. Elevated sdLDL levels are associated with metabolic syndrome and an 'atherogenic lipoprotein profile', and are a strong, independent predictor of cardiovascular disease. Elevated levels of LDL arise from multiple sources. A major factor is sedentary lifestyle with a diet high in saturated fat. Insulin-resistance and pre-diabetes have also been implicated, as has genetic predisposition. Measurement of sdLDL allows the clinician to get a more comprehensive picture of lipid risk factors and tailor treatment accordingly. Reducing LDL levels will reduce the risk of CVD and MI.

SEX: Male

Non HDL Cholesterol - Adult treatment panel ATP III suggested the addition of Non-HDL Cholesterol as an indicator of all atherogenic lipoproteins (mainly LDL and VLDL).

NICE guidelines recommend Non-HDL Cholesterol measurement before initiating lipid lowering therapy. It has also been shown to be a better marker of risk in both primary and secondary prevention studies.

Results of Lipids should always be interpreted in conjunction with the patient's medical history, clinical presentation and other findings.

NON FASTING LIPID PROFILE includes Total Cholesterol, HDL Cholesterol and calculated non-HDL Cholesterol. It does not include triglycerides and may be best used in patients for whom fasting is difficult.

LIVER FUNCTION PROFILE, SERUM-

LIVER FUNCTION PROFILE

Bilirubin is a yellowish pigment found in bile and is a breakdown product of normal heme catabolism. Bilirubin is excreted in bile and urine, and elevated levels may give yellow discoloration in jaundice. Elevated levels results from increased bilirubin production (eg, hemolysis and ineffective erythropoiesis), decreased bilirubin excretion (eg, obstruction and hepatitis), and abnormal bilirubin metabolism (eg, hereditary and neonatal jaundice). Conjugated (direct) bilirubin is elevated more than unconjugated (indirect) bilirubin in Viral hepatitis, Drug reactions, Alcoholic liver disease Conjugated (direct) bilirubin is also elevated more than unconjugated (indirect) bilirubin when there is some kind of blockage of the bile ducts like in Gallstones getting into the bile ducts, tumors &Scarring of the bile ducts. Increased unconjugated (indirect) bilirubin may be a result of Hemolytic or pernicious anemia, Transfusion reaction & a common metabolic condition termed Gilbert syndrome, due to low levels of the enzyme that attaches sugar molecules to bilirubin.
AST is an enzyme found in various parts of the body. AST is found in the liver, heart, skeletal muscle, kidneys, brain, and red blood cells, and it is commonly measured

clinically as a marker for liver health. AST levels increase during chronic viral hepatitis, blockage of the bile duct, cirrhosis of the liver, liver cancer, kidney failure, hemolytic anemia, pancreatitis, hemochromatosis. AST levels may also increase after a heart attack or strenuous activity. ALT test measures the amount of this enzyme in the blood. ALT is found mainly in the liver, but also in smaller amounts in the kidneys, heart, muscles, and pancreas. It is commonly measured as a part of a diagnostic evaluation of hepatocellular injury, to determine liver health.AST levels increase during acute hepatitis, sometimes due to a viral infection, ischemia to the liver, chronic hepatitis, obstruction of bile ducts, cirrhosis.

ALP is a protein found in almost all body tissues. Tissues with higher amounts of ALP include the liver, bile ducts and bone. Elevated ALP levels are seen in Biliary obstruction, Osteoblastic bone tumors, osteomalacia, hepatitis, Hyperparathyroidism, Leukemia, Lymphoma, 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 pancreas. 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 NITROGEN-

Causes of Increased levels Pre renal

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

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



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CLIENT CODE: C000138383

CLIENT'S NAME AND ADDRESS: ACROFEMI HEALTHCARE LTD (MEDIWHEEL)

F-703, LADO SARAI, MEHRAULI

SOUTH WEST DELHI **NEW DELHI 110030 DELHI INDIA**

8800465156

SRL Ltd

24 SCO, SECTOR 11 D CHANDIGARH, 160011 PUNJAB, INDIA

Tel: 9111591115, Fax: CIN - U74899PB1995PLC045956

PATIENT NAME: SUNIL KUMAR 528408607650 PATIENT ID: SUNIM11068580

0080VG007192 AGE: 37 Years ACCESSION NO: SEX: Male ABHA NO:

DRAWN: RECEIVED: 16/07/2022 09:43 REPORTED: 16/07/2022 16:54

REFERRING DOCTOR: SFLF CLIENT PATIENT ID:

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

• 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 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-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.

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

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. Urinary protein excretion can also be temporarily elevated by strenuous exercise, orthostatic proteinuria, dehydration, urinary 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 can lead to presence of ketones in urine. Ketones can also be seen in starvation, frequent vomiting, pregnancy and strenuous exercise.

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 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

THYROID PANEL, SERUM-

Triiodothyronine 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

TOTAL T4 (µg/dL) Levels in TSH3Ġ TOTAL T3 Pregnancy (uIU/mL) (na/dL)



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6.6 - 12.4 6.6 - 15.5 0.1 - 2.5 0.2 - 3.0 First Trimester 81 - 190 100 - 260 100 - 260 2nd Trimester 3rd Trimester 6.6 - 15.5 0.3 - 3.0 Below mentioned are the guidelines for age related reference ranges for T3 and T4. $\mathsf{T3}$

(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.

1. Bortis C.A., Ashwood E. R. Bruis D.E. Tellz textbook of Clinical Cremistry and Molecular Diagnostics, 4th Edition.

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

3. Behrman R.E. Kilegman R.M., Jenson H. B. Nelson Text Book of Pediatrics, 17th Edition

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.

End Of Report

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

Dr. Pranjali Vasisht **LAB HEAD**

saculit



