

CLIENT'S NAME AND ADDRESS:

ACROFEMI HEALTHCARE LTD (MEDI WHEEL)

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: ANIL NAMDEV SALUNKHE

PATIENT ID:

REPORTED: 27/10/2022 14:26:23

ANILM221076181

ACCESSION NO: 0181VJ001042 AGE: 46 Years

SEX: Male

ABHA NO:

DRAWN:

RECEIVED: 22/10/2022 08:59:43

REFERRING DOCTOR: SELF CLIENT PATIENT ID:

Test Report Status	<u>Final</u>	Results	Biological Reference	Interval Units
MEDI WHEEL EILL BO	NEW HEALTH CHECK HE	ABOVE 40 MALE		
	DDY HEALTH CHECK UP	ABOVE 40 MALE		
BLOOD COUNTS,EDTA	MHOLE BLOOD			
HEMOGLOBIN		15.8	13.0 - 17.0	g/dL
METHOD : SLS-HEMOGLOBII		E 46	45.55	217.1
RED BLOOD CELL COUN		5.16	4.5 - 5.5	mil/μL
METHOD : HYDRODYNAMIC F		7.60	4.0 10.0	Hoov (u)
WHITE BLOOD CELL CO		7.62	4.0 - 10.0	thou/µL
	LOW CYTOMETRY	180	150 - 410	thou/µL
PLATELET COUNT METHOD: HYDRODYNAMIC F	OCHEING BY DO DETECTION	100	130 - 410	αιοα/με
RBC AND PLATELET I				
	MDICES	46.7	40.0 50.0	%
HEMATOCRIT	SE HEIGHT DETECTION METHOD	40.7	40.0 - 50.0	70
MEAN CORPUSCULAR V		90.5	83.0 - 101.0	fL
METHOD : CALCULATED FROM		90.5	03.0 101.0	IL.
MEAN CORPUSCULAR H		30.6	27.0 - 32.0	pg
METHOD : CALCULATED FROM		00.0	27.0 02.0	P9
MEAN CORPUSCULAR H		33.8	31.5 - 34.5	g/dL
CONCENTRATION				<i>31</i>
METHOD : CALCULATED FROI	M THE HGB & HCT			
MENTZER INDEX		17.5		
RED CELL DISTRIBUTIO		13.3	11.6 - 14.0	%
	M RBC SIZE DISTRIBUTION CURVE			
MEAN PLATELET VOLUM	- -	10.1	6.8 - 10.9	fL
	M PLATELET COUNT & PLATELET H	EMATOCRIT		
WBC DIFFERENTIAL	COUNT - NLR			
NEUTROPHILS		65	40 - 80	%
METHOD : FLOW CYTOMETRY				
ABSOLUTE NEUTROPHII		4.92	2.0 - 7.0	thou/μL
METHOD : FLOW CYTOMETRY	WITH LIGHT SCATTERING			
LYMPHOCYTES		29	20 - 40	%
METHOD : FLOW CYTOMETRY				
ABSOLUTE LYMPHOCYT		2.19	1.0 - 3.0	thou/µL
METHOD : FLOW CYTOMETRY		2.2		
NEUTROPHIL LYMPHOC	TIE KATIO (NLK)	2.3		
EOSINOPHILS		1	1 - 6	%



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METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING				
ABSOLUTE EOSINOPHIL COUNT	0.08		0.02 - 0.50	thou/µL
METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING	2.22			,,
MONOCYTES	5		2 - 10	%
METHOD: FLOW CYTOMETRY WITH LIGHT SCATTERING				
ABSOLUTE MONOCYTE COUNT	0.40		0.2 - 1.0	thou/µL
METHOD: FLOW CYTOMETRY WITH LIGHT SCATTERING				
DIFFERENTIAL COUNT PERFORMED ON:	EDTA SMEAR			
MORPHOLOGY				
RBC	NORMOCYTIC N	IORMOCHRO	OMIC	
WBC	NORMAL MORPHOLOGY			
METHOD: MICROSCOPIC EXAMINATION	Hora Da Hora			
PLATELETS	ADEQUATE			
ERYTHROCYTE SEDIMENTATION RATE (ESR)	-			
E.S.R	21	High	< 15	mm at 1 hr
GLYCOSYLATED HEMOGLOBIN(HBA1C), EDT BLOOD	A WHOLE			
HBA1C	5.7		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) METHOD : CALCULATED PARAMETER	116.9	High	< 116.0	mg/dL
GLUCOSE FASTING, FLUORIDE PLASMA				
FBS (FASTING BLOOD SUGAR)	118	High	Normal 75 - 99 Pre-diabetics: 100 - 125 Diabetic: > or = 126	mg/dL
METHOD: ENZYMATIC REFERENCE METHOD WITH HEXOKINAS	GE .			
GLUCOSE, POST-PRANDIAL, PLASMA				
PPBS(POST PRANDIAL BLOOD SUGAR)	99		70 - 139	mg/dL
METHOD: ENZYMATIC REFERENCE METHOD WITH HEXOKINAS	SE .			



CORONARY RISK PROFILE, SERUM



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CHOLESTEROL, TOTAL	184		Desirable cholesterol level < 200 Borderline high cholesterol 200 - 239 High cholesterol > / = 240	mg/dL
METHOD: ENZYMATIC COLORIMETRIC ASSAY	169	Uiah	Normali < 150	ma/dl
TRIGLYCERIDES	169	nigii	Normal: < 150 Borderline high: 150 - 199 High: 200 - 499 Very High: >/= 500	mg/dL
METHOD : ENZYMATIC COLORIMETRIC ASSAY				
HDL CHOLESTEROL	32	Low	Low HDL Cholesterol <40	mg/dL
METHOD : ENZYMATIC, COLORIMETRIC			High HDL Cholesterol >/= 60)
CHOLESTEROL LDL	118	High	Adult levels: Optimal < 100 Near optimal/above optimal: 1 129 Borderline high: 130-159 High: 160-189 Very high: = 190	mg/dL L00-
METHOD: ENZYMATIC COLORIMETRIC ASSAY			very high . – 190	
NON HDL CHOLESTEROL	152	High	Desirable : < 130 Above Desirable : 130 -159 Borderline High : 160 - 189 High : 190 - 219 Very high : > / = 220	mg/dL
CHOL/HDL RATIO	5.8	High	Low Risk: 3.3 - 4.4 Average Risk: 4.5 - 7.0 Moderate Risk: 7.1 - 11.0 High Risk: > 11.0	
LDL/HDL RATIO	3.7	High	0.5 - 3.0 Desirable/Low Risk 3.1 - 6.0 Borderline/Moderate >6.0 High Risk	Risk
VERY LOW DENSITY LIPOPROTEIN	33.8	High	< OR = 30.0	mg/dL
LIVER FUNCTION PROFILE, SERUM				
BILIRUBIN, TOTAL METHOD: COLORIMETRIC DIAZO	0.92		Upto 1.2	mg/dL
BILIRUBIN, DIRECT	0.30		< 0.30	mg/dL
BILIRUBIN, INDIRECT	0.62		0.1 - 1.0	mg/dL



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TOTAL PROTEIN	7.7	6.0 - 8.0	g/dL	
METHOD : COLORIMETRIC			<i>3</i> ,	
ALBUMIN METHOD: COLORIMETRIC	4.4	3.97 - 4.94	g/dL	
GLOBULIN	3.3	2.0 - 3.5	g/dL	
ALBUMIN/GLOBULIN RATIO	1.3	1.0 - 2.1	RATIO	
	29			
ASPARTATE AMINOTRANSFERASE (AST/SGOT) METHOD: UV ABSORBANCE	29	< OR = 50	U/L	
ALANINE AMINOTRANSFERASE (ALT/SGPT) METHOD: UV ABSORBANCE	31	< OR = 50	U/L	
ALKALINE PHOSPHATASE	51	40 - 129	U/L	
METHOD : COLORIMETRIC				
GAMMA GLUTAMYL TRANSFERASE (GGT)	30	0 - 60	U/L	
METHOD : ENZYMATIC, COLORIMETRIC				
LACTATE DEHYDROGENASE	192	125 - 220	U/L	
METHOD : UV ABSORBANCE				
BLOOD UREA NITROGEN (BUN), SERUM				
BLOOD UREA NITROGEN	7	6 - 20	mg/dL	
METHOD : ENZYMATIC ASSAY				
CREATININE, SERUM				
CREATININE	0.73	0.7 - 1.2	mg/dL	
METHOD : COLORIMETRIC				
BUN/CREAT RATIO				
BUN/CREAT RATIO	9.59	8.0 - 15.0		
URIC ACID, SERUM				
URIC ACID	6.0	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				
ALBUMIN	4.4	3.97 - 4.94	g/dL	
METHOD : COLORIMETRIC				
GLOBULIN				
GLOBULIN	3.3	2.0 - 3.5	g/dL	



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ELECTROLIVES (NA (V/OL) CERUM			
ELECTROLYTES (NA/K/CL), SERUM	120	100 145	
SODIUM	138	136 - 145	mmol/L
POTASSIUM	3.80	3.5 - 5.1	mmol/L
CHLORIDE	101	98 - 107	mmol/L
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			
CHEMICAL EXAMINATION, URINE			
PH	6.0	4.7 - 7.5	
METHOD: DOUBLE INDICATOR PRINCIPLE			
PROTEIN	NOT DETECTED	NOT DETECTED	
METHOD: TETRA BROMOPHENOL BLUE/SULFOSALICYLIC ACID			
GLUCOSE	NOT DETECTED	NOT DETECTED	
METHOD: GLUCOSE OXIDASE PEROXIDASE			
KETONES	NOT DETECTED	NOT DETECTED	
METHOD: NITROPRUSSIDE REACTION			
BLOOD	NOT DETECTED	NOT DETECTED	
METHOD: PEROXIDASE			
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	NOT DETECTED	NOT DETECTED	
LEUKOCYTE ESTERASE	NOT DETECTED	NOT DETECTED	
MICROSCOPIC EXAMINATION, URINE			
PUS CELL (WBC'S)	1-2	0-5	/HPF
METHOD: MICROSCOPIC EXAMINATION			
EPITHELIAL CELLS	0-1	0-5	/HPF
METHOD: MICROSCOPIC EXAMINATION	NOT DETECTE	WAT DET	
ERYTHROCYTES (RBC'S)	NOT DETECTED	NOT DETECTED	/HPF
METHOD: MICROSCOPIC EXAMINATION	NOT DETECTE		
CASTS	NOT DETECTED		





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Test Report Status Results Biological Reference Interval Units <u>Final</u> METHOD: MICROSCOPIC EXAMINATION **CRYSTALS** NOT DETECTED METHOD: MICROSCOPIC EXAMINATION NOT DETECTED NOT DETECTED BACTERIA METHOD: MICROSCOPIC EXAMINATION NOT DETECTED NOT DETECTED YEAST THYROID PANEL, SERUM 100.0 80 - 200 ng/dL METHOD: ELECTROCHEMILUMINESCENCE T4 5.15 5.1 - 14.1μg/dL METHOD: ELECTROCHEMILUMINESCENCE TSH 3RD GENERATION 2.480 0.27 - 4.2μIU/mL METHOD: ELECTROCHEMILUMINESCENCE ABO GROUP & RH TYPE, EDTA WHOLE BLOOD ABO GROUP TYPE O

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

ECG WITHIN NORMAL LIMITS

MEDICAL HISTORY

RELEVANT PRESENT HISTORY CONIUNCTINITIS SINCE TODAY MORNING.

RELEVANT PAST HISTORY NOT SIGNIFICANT

RELEVANT PERSONAL HISTORY MARRIED / 2 CHILD / MIXED DIET / NO ALLERGIES / NO SMOKING /

OCC. ALCOHOL.

RELEVANT FAMILY HISTORY NOT SIGNIFICANT HISTORY OF MEDICATIONS NOT SIGNIFICANT

ANTHROPOMETRIC DATA & BMI

HEIGHT IN METERS 1.71 mts WEIGHT IN KGS. 103 Kgs



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ВМІ	35	BMI & Weight Status as follows: kg/sqmts 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	OBESE	

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

BRUIT

RESPIRATORY RATE NORMAL

CARDIOVASCULAR SYSTEM

BP 150/90 MM HG 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





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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 CONJUNCTIVITIS CONGESTION BOTH EYES

EYELIDS NORMAL
EYE MOVEMENTS NORMAL
CORNEA NORMAL

DISTANT VISION RIGHT EYE WITHOUT GLASSES REDUCED VISUAL ACUITY 6/12
DISTANT VISION LEFT EYE WITHOUT GLASSES REDUCED VISUAL ACUITY 6/12

NEAR VISION RIGHT EYE WITHOUT GLASSES WITHIN NORMAL LIMIT NEAR VISION LEFT EYE WITHOUT GLASSES WITHIN NORMAL LIMIT

COLOUR VISION NORMAL

SUMMARY

RELEVANT HISTORY NOT SIGNIFICANT
RELEVANT GP EXAMINATION FINDINGS OBESE: - BMI 35



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REMARKS / RECOMMENDATIONS ADVICE:-

1)OPHTHALMOLOGY CONSULT FOR CONJUNCTIVITIS

2) HEPATOLOGY CONSULT ON VIEW OF USG FINDINGS. 3)BP MONITORING FOR 5 DAYS. IF PERSISTENTLY HIGH, WILL REQUIRE

EVALUATION BY PHYSICIAN.BP MONITORING AND FOLLOW UP. 4)ADD YOGA, PRANAYAM MEDITATION TO DAILY ROUTINE.

5) WEIGHT LOSS -STRICT LOW SALT, LOW FAT, LOW CARBOHYDRATE,

HIGH FIBRE DIET.

6) REGULAR EXERCISE, REGULAR WALK FOR 30-40 MIN DAILY.

7) REPEAT BLOOD SUGAR, LIPID PROFILE AFTER 3 MONTHS OF DIET

AND EXERCISE.



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MEDI WHEEL FULL BODY HEALTH CHECK UP ABOVE 40 MALE

ULTRASOUND ABDOMEN

ULTRASOUND ABDOMEN

HEPATOMEGALY WITH GRADE II FATTY LIVER.

Interpretation(s)
BLOOD COUNTS,ED TA WHOLE BLOODThe 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-

The optimal threshold of 3.3 for NLR showed a prognostic possibility of dinical 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.

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 dear fluid (plasma) that are present at the top portion of the tube after one hour. Nowadays fully automated instruments are available to measure ESR.

ESR is not diagnostic; it is a non-specific test that may be elevated in a number of different conditions. It provides general information about the presence of an inflammatory condition.CRP is superior to ESR because it is more sensitive and reflects a more rapid change.

TEST INTERPRETATION

Increase in: Infections, Vasculities, Inflammatory arthritis, Renal disease, Anemia, Malignancies and plasma cell dyscrasias, Acute allergy Tissue injury, Pregnancy,

Estrogen medication, Aging.
Finding a very accelerated ESR(>100 mm/hour) in patients with ill-defined symptoms directs the physician to search for a systemic disease (Paraproteinemias, Disseminated malignancies, connective tissue disease, severe infections such as bacterial endocarditis).

In pregnancy BRI in first trimester is 0-48 mm/hr(62 if anemic) and in second trimester (0-70 mm/hr(95 if anemic). ESR returns to normal 4th week post partum. Decreased in: Polycythermia vera, Sickle cell anemia

False elevated ESR: Increased fibrinogen, Drugs(Vitamin A, Dextran etc), Hypercholesterolemia

False Decreased: Poikilocytosis, (Sickle Cells, spherocytes), Microcytosis, Low fibrinogen, Very high WBC counts, Drugs (Quinine,

salicylates)

1. Nathan and Oski's Haematology of Infancy and Childhood, 5th edition; 2. Paediatric reference intervals. AACC Press, 7th edition. Edited by S. Soldin; 3. The reference for the adult reference range is "Practical Haematology by Dacie and Lewis, 10th edition.
GLYCOSYLATED HEMOGLOBIN(HBA1C), EDTA WHOLE BLOOD-**Used For:**

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

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

1.eAG (Estimated average glucose) converts percentage HbA1c to md/dl, to compare blood glucose levels.

2. eAG gives an evaluation of blood glucose levels for the last couple of months.

- 3. eAG is calculated as eAG (mg/dl) = 28.7 * HbA1c 46.7

HbA1c Estimation can get affected due to:

I.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.

II.Vitamin C & E are reported to falsely lower test results (possibly by inhibiting glycation of hemoglobin.
III.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.

IV.Interference of hemoglobinopathies in HbA1c estimation is seen in

a. Homozygous hemoglobinopathy. Fructosamine is recommended for testing of HbA1c.





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PATIENT NAME: ANIL NAMDEV SALUNKHE

PATIENT ID: ANILM221076181

AGE: 46 Years SEX: Male ABHA NO: ACCESSION NO : 0181VJ001042

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b.Heterozygous state detected (D10 is corrected for HbS & HbC trait.)

c.HbF > 25% on alternate paltform (Boronate affinity chromatography) is recommended for testing of HbA1c.Abnormal Hemoglobin electrophoresis (HPLC method) is

recommended for detecting a hemoglobinopathy
GLUCOSE FASTING,FLUORIDE PLASMA-TEST DESCRIPTION

Normally, the glucose concentration in extracellular fluid is closely regulated so that a source of energy is readily available to tissues and sothat no glucose is excreted in the

Increased in

Diabetes mellitus, Cushina's syndrome (10 - 15%), chronic pancreatitis (30%), Drugs; corticostergids, phenytoin, estrogen, thiazides,

Decreased in

Pancreatic islet cell disease with increased insulin,insulinoma, adrenocortical insufficiency, hypopituitarism, diffuse liver disease, malignancy (adrenocortical, stomach,fibrosarcoma), infant of a diabetic mother, enzyme deficiency diseases(e.g., galactosemia),Drugs-insulin, ethanol, propranolol; sulfonylureas, tolbutamide, and other oral hypoglycemic agents.

NOTE:

While random serum glucose levels correlate with home glucose monitoring results (weekly mean capillary glucose values), there is wide fluctuation within individuals. Thus,

GLUCOSE, POST-PRANDIAL, PLASMA-High fasting glucose level in comparison to post prandial glucose level in comparison to post prandial glucose level may be seen due to effect of Oral Hypoglycaemics & Insulin treatment, Renal Glyosuria, Glycaemic index & response to food consumed, Alimentary Hypoglycemia, Increased insulin response & sensitivity etc.

GLUCOSE, POST-PRANDIAL, PLASMA-High fasting glucose level in comparison to post prandial glucose level may be seen due to effect of Oral Hypoglycaemics & Insulin

treatment, Renal Glyosuria, Glycaemic index & response to food consumed, Alimentary Hypoglycemia, Increased insulin response & sensitivity etc. Additional test HbA1c LIVER FUNCTION PROFILE, SERUM-

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 enythropoiesis), 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.

actacles sugnifications to bilinguistics.

AST is an enzyme found in various parts of the body. AST is found in the liver, heart, skeletal musde, 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,musdes, 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, osteomaladia, 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, biliary 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

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)
 Musde problems, such as breakdown of muscle fibers

- Problems during pregnancy, such as seizures (eclampsia)), or high blood pressure caused by pregnancy (preedampsia)

Lower than normal level may be due to:
• Myasthenia Gravis

- Múscular dystrophy

URIC ACID, ŚERUM-

Causes of Increased levels

Dietary
• High Protein Intake.





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Prolonged Fasting,

Rapid weight loss.

Gout

Lesch nyhan syndrome.

Type 2 ĎM.

Metabolic syndrome.

Causes of decreased levels

- Low Zinc Intake
- OCP's
- Multiple Scienosis

Nutritional tips to manage increased Uric acid levels

- Drink plenty of fluids
 Limit animal proteins
- High Fibre foodsVit 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.

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 dearance, malnutrition 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 réspiratory acidosís, salt-losing nephritis, metabolic alkalosis, congestive heart failure, Addisónian crisis, certain types of metabolic acidosis, persistent gastric secretion and

prolonged vomiting,
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, 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 m edications.

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-Triodothyronine 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 (T5H), which is released from the pituitary gland. Elevated concentrations of T3, and T4 in the blood inhibit the production of T5H.

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 TSH3G TOTAL T3





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(μΙU/mL) 0.1 - 2.5 0.2 - 3.0 0.3 - 3.0 (µg/dL) 6.6 - 12.4 6.6 - 15.5 (ng/dL) 81 - 190 100 - 260 100 - 260 Pregnancy First Trimester 2nd Trimester 6.6 - 15.5 3rd Trimester Below mentioned are the guidelines for age related reference ranges for T3 and T4.

(ng/dL) (µg/dL) 1-3 day: 8.2 - 19.9 1 Week: 6.0 - 15.9 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.

Burtis C.A., Ashwood E. R. Bruns D.E. Teitz textbook of Clinical Chemistry and Molecular Diagnostics, 4th Edition.
 Gowenlock A.H. Varley"'s Practical Clinical Biochemistry, 6th Edition.
 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.

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.

End Of Report

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





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