

CLIENT'S NAME AND ADDRESS : ACROFEMI HEALTHCARE LTD (MEDIWHEEL) F-703, F-703, LADO SARAI, MEHRAULI SOUTH WEST DELHI NEW DELHI 110030 DELHI INDIA 9800465156 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 : NEHA GUPTA			PATIENT ID : NEHAF091291181
ACCESSION NO :	0181VD000919	AGE : 30 Years SEX : Female	
DRAWN :		RECEIVED : 14/04/2022 11:08	REPORTED : 15/04/2022 14:58
REFERRING DOCT	OR: SELF		CLIENT PATIENT ID :

Test Report Status Final Results Biological Reference Interval Units
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MEDI WHEEL FULL BODY HEALTH CHECKUP BELOW 40FEMALE

E	BLOOD COUNTS, EDTA WHOLE BLOOD				
F	IEMOGLOBIN	11.8	Low	12.0 - 15.0	g/dL
	METHOD : SLS- HEMOGLOBIN DETECTION METHOD				
R	ED BLOOD CELL COUNT	3.95		3.8 - 4.8	mil/µL
	METHOD : HYDRODYNAMIC FOCUSING BY DC DETECTION				
۷	VHITE BLOOD CELL COUNT	8.40		4.0 - 10.0	thou/µL
	METHOD : FLUORESCENCE FLOW CYTOMETRY				
Ρ	LATELET COUNT	278		150 - 410	thou/µL
	METHOD : HYDRODYNAMIC FOCUSING BY DC DETECTION				
F	BC AND PLATELET INDICES				
F	IEMATOCRIT	36.0		36.0 - 46.0	%
	METHOD : CUMULATIVE PULSE HEIGHT DETECTION METHOD				
Ν	IEAN CORPUSCULAR VOL	91.1		83.0 - 101.0	fL
	METHOD : CALCULATED FROM RBC & HCT				
Ν	IEAN CORPUSCULAR HGB.	29.9		27.0 - 32.0	pg
	METHOD : CALCULATED FROM THE RBC & HGB				
	IEAN CORPUSCULAR HEMOGLOBIN	32.8		31.5 - 34.5	g/dL
	METHOD : CALCULATED FROM THE HGB & HCT				
Ν	IENTZER INDEX	23.1			
R	ED CELL DISTRIBUTION WIDTH	12.5		11.6 - 14.0	%
	METHOD : CALCULATED FROM RBC SIZE DISTRIBUTION CURVE				
Ν	IEAN PLATELET VOLUME	11.9	High	6.8 - 10.9	fL
	METHOD : CALCULATED FROM PLATELET COUNT & PLATELET HEMAT	OCRIT			
V	VBC DIFFERENTIAL COUNT - NLR				
S	EGMENTED NEUTROPHILS	57		40 - 80	%
	METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING				
A	BSOLUTE NEUTROPHIL COUNT	4.79		2.0 - 7.0	thou/µL
	METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING				
L	YMPHOCYTES	31		20 - 40	%
	METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING				
Α	BSOLUTE LYMPHOCYTE COUNT	2.59		1.0 - 3.0	thou/µL
	METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING				
Ν	IEUTROPHIL LYMPHOCYTE RATIO (NLR)	1.9			
E	OSINOPHILS	7	High	1 - 6	%
	METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING				

METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING







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ABSOLUTE EOSINOPHI	_ COUNT	0.62	High	0.02 - 0.50	thou/µL
METHOD : FLOW CYTOMETRY	WITH LIGHT SCATTERING				
MONOCYTES		5		2 - 10	%
METHOD : FLOW CYTOMETRY		0.40			
ABSOLUTE MONOCYTE		0.42		0.2 - 1.0	thou/µL
DIFFERENTIAL COUNT		EDTA SMEAR			
MORPHOLOGY					
RBC		NORMOCYTIC N		DMIC	
WBC		NORMAL MORPH	IOLOGY		
METHOD : MICROSCOPIC EX	AMINATION				
PLATELETS		ADEQUATE			
ERYTHRO SEDIMENT	-				
SEDIMENTATION RATE		21	High	0 - 20	mm at 1 hr
METHOD : WESTERGREN ME					
GLUCOSE, FASTING,					
GLUCOSE, FASTING, PL		86		74.0 - 106.0	mg/dL
METHOD : GLUCOSE OXIDAS					
	OGLOBIN, EDTA WHOLE				
GLYCOSYLATED HEMOC	GLOBIN (HBA1C)	5.1		Non-diabetic: < 5.7 Pre-diabetics: 5.7 - 6.4 Diabetics: > or = 6.5 ADA Target: 7.0 Action suggested: > 8.0	%
METHOD : HPLC	_				<i>,</i>
MEAN PLASMA GLUCOS		99.7		< 116.0	mg/dL
METHOD : CALCULATED PAR					
GLUCOSE, POST-PRA		107		74 140	
GLUCOSE, POST-PRANE	•	107		74 - 140	mg/dL
METHOD : GLUCOSE OXIDAS	。□ DFILE (LIPID PROFILE),	CEDUM			
	FILE (LIFID PROFILE),				ma a /dl
CHOLESTEROL		114		< 200 Desirable 200 - 239 Borderline High >/= 240 High	mg/dL
METHOD : CHOLESTEROL OX	IDASE				
TRIGLYCERIDES		70		Normal: <150 Borderline high: 150 - 199 High: 200 - 499 Very high: > or = 500	mg/dL
METHOD , ENZYMATIC ACCA	V				

METHOD : ENZYMATIC ASSAY







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HDL CHOLESTEROL		40		< 40 Low >/=60 High	mg/dL
METHOD : DIRECT- NON IMM	IUNOLOGICAL			27=00 mgn	
DIRECT LDL CHOLESTE		64		< 100 Optimal 100 - 129 Near or above optima 130 - 159 Borderline High 160 - 189 High >/= 190 Very High	mg/dL al
METHOD : ENZYMATIC ASSA					
NON HDL CHOLESTERO		74			mg/dL
METHOD : CALCULATED PARA	AMETER				
CHOL/HDL RATIO	AMETED	2.9	Low	3.3- 4.4 Low Risk 4.5 -7.0 Average Risk 7.1 -11.0 Moderate Risk > 11.0 High Risk	
LDL/HDL RATIO		1.6		0.5 - 3.0 Desirable/Low Risk	
		1.0		3.1 - 6.0 Borderline/Moderate R >6.0 High Risk	lisk
METHOD : CALCULATED PARA		14.0		10.25	
VERY LOW DENSITY LIP METHOD : CALCULATED PARA		14.0		10 - 35	mg/dL
LIVER FUNCTION PRO					
BILIRUBIN, TOTAL		0.37		0.2 - 1.3	mg/dL
METHOD : DIPHYLLINE DIAZO	ONIUM SALTS	0.57		0.2 1.5	ing/ac
BILIRUBIN, DIRECT		0.10		0.0 - 0.3	mg/dL
METHOD : DIPHYLLINE DIAZO	ONIUM SALTS				
BILIRUBIN, INDIRECT		0.27		0.0 - 1.1	mg/dL
METHOD : DIPHYLLINE DIAZO	ONIUM SALTS				
TOTAL PROTEIN		7.1		6.3 - 8.3	g/dL
ALBUMIN		4.2		3.5 - 5.0	g/dL
GLOBULIN		2.9		2.0 - 3.5	g/dL
ALBUMIN/GLOBULIN RA	TIO	1.5		1.0 - 2.0	RATIO
ASPARTATE AMINOTRA	NSFERASE (AST/SGOT)	28		14 - 36	U/L
ALANINE AMINOTRANS	FERASE (ALT/SGPT)	24		< 35.0	U/L
ALKALINE PHOSPHATAS	SE	73		38 - 126	U/L
GAMMA GLUTAMYL TRA	NSFERASE (GGT)	11	Low	12 - 43	U/L
LACTATE DEHYDROGEN	ASE	204		120 - 246	U/L
SERUM BLOOD UREA	NITROGEN				
BLOOD UREA NITROGE		10		7.0 - 17.0	mg/dL







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METHOD : UREASE WITH INDICATOR	R DYE				
CREATININE, SERUM CREATININE		0.63		0.52 - 1.04	ma/dl
METHOD : ENZYMETIC IDMS		0.05		0.32 - 1.04	mg/dL
BUN/CREAT RATIO					
BUN/CREAT RATIO		15.87			
URIC ACID, SERUM		10107			
URIC ACID		3.1		2.5 - 6.2	mg/dL
METHOD : URICASE UV		5.1		2.5 0.2	ilig/uL
TOTAL PROTEIN, SERUM					
TOTAL PROTEIN		7.1		6.3 - 8.30	g/dL
METHOD : BIURET, END POINT		,12			9,42
ALBUMIN, SERUM					
ALBUMIN		4.2		3.5 - 5.0	g/dL
METHOD : BCG DYE BINDING METHO	DD				5, -
GLOBULIN					
GLOBULIN		2.9		2.0 - 3.5	g/dL
METHOD : CALCULATED PARAMETER					
ELECTROLYTES (NA/K/CL)), SERUM				
SODIUM		135 L	Low	137 - 145	mmol/L
METHOD : ION SELECTIVE ELECTRON	DE TECHNOLOGY				
POTASSIUM		4.3		3.6 - 5.0	mmol/L
METHOD : ION SELECTIVE ELECTRON	DE TECHNOLOGY				
CHLORIDE		99		98 - 107	mmol/L
METHOD : ION SELECTIVE ELECTRON	DE TECHNOLOGY				
URINALYSIS					
COLOR		PALE YELLOW			
METHOD : VISUAL INSPECTION					
APPEARANCE		CLEAR			
METHOD : VISUAL INSPECTION		C D			
		6.0		4.7 - 7.5	
METHOD : DOUBLE INDICATOR PRIN	ICIPLE	1 020		1 002 1 025	
SPECIFIC GRAVITY METHOD : IONIC CONCENTRATION M	METHOD	1.030		1.003 - 1.035	
GLUCOSE		NOT DETECTED		NOT DETECTED	
METHOD : GLUCOSE OXIDASE PERO	XIDASE				
PROTEIN	-	NOT DETECTED		NOT DETECTED	







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METHOD : TETRA BROMOPHENOL BLUE/SULFOSALICYLIC ACID				
KETONES	NOT DETECTED	NOT DETECTED		
METHOD : NITROPRUSSIDE REACTION	NOT DETECTED	NOT DETECTED		
BLOOD	NOT DETECTED	NOT DETECTED		
METHOD : PEROXIDASE				
UROBILINOGEN	NORMAL	NORMAL		
METHOD : MODIFIED EHRLICH REACTION				
NITRITE	NOT DETECTED	NOT DETECTED		
METHOD : 1,2,3,4-TETRAHYDROBENZO(H)QUINOLIN-3-OL				
PUS CELL (WBC'S)	2-3	0-5	/HPF	
METHOD : MICROSCOPIC EXAMINATION				
EPITHELIAL CELLS	2-3	0-5	/HPF	
METHOD : MICROSCOPIC EXAMINATION				
ERYTHROCYTES (RBC'S)	NOT DETECTED	NOT DETECTED	/HPF	
METHOD : MICROSCOPIC EXAMINATION				
CASTS	NOT DETECTED			
METHOD : MICROSCOPIC EXAMINATION				
CRYSTALS	NOT DETECTED			
METHOD : MICROSCOPIC EXAMINATION				
BACTERIA	NOT DETECTED	NOT DETECTED		
METHOD : MICROSCOPIC EXAMINATION				
THYROID PANEL, SERUM				
Т3	94.9	58 - 159	ng/dL	
METHOD : CHEMILUMINESCENT MICROPARTICLE IMMUNO ASSAY				
Τ4	10.15	4.87 - 11.71	µg/dL	
METHOD : CHEMILUMINESCENT MICROPARTICLE IMMUNO ASSAY				
TSH 3RD GENERATION	1.277	0.350 - 4.940	µIU/mL	
METHOD : CHEMILUMINESCENT MICROPARTICLE IMMUNO ASSAY				
PAPANICOLAOU SMEAR				
TEST METHOD	CONVENTIONAL GYNEC CYTOLOGY			
METHOD : MICROSCOPIC EXAMINATION				
SPECIMEN TYPE	P - 232/22 TWO UNSTAINED CERVIC	AL SMEARS RECEIVED		
METHOD : MICROSCOPIC EXAMINATION				
REPORTING SYSTEM	2014 BETHESDA SYSTEM FOR REPORTING CERVICAL CYTOLOGY			
SPECIMEN ADEQUACY	SATISFACTORY			
METHOD : PAP STAIN & MICROSCOPIC EXAMINATION				







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MICROSCOPY	INTERMEDIATE SQUAMO	' SUPERFICIAL SQUAMOUS CELLS US CELLS AND FEW CLUSTERS O THE BACKGROUND OF POLYMOR	F
METHOD : PAP STAIN			
INTERPRETATION / RESULT	NEGATIVE FOR INTRAEPI	THELIAL LESION OR MALIGNANCY	(
METHOD : PAP STAIN & MICROSCOPIC EXAMINATION			
STOOL: OVA & PARASITE			
COLOUR	BROWN		
METHOD : VISUAL			
CONSISTENCY	WELL FORMED		
METHOD : VISUAL			
ODOUR	FAECAL		
METHOD : PHYSICAL			
MUCUS	NOT DETECTED	NOT DETECTED	
METHOD : VISUAL			
VISIBLE BLOOD	ABSENT	ABSENT	
METHOD : VISUAL			
POLYMORPHONUCLEAR LEUKOCYTES	1-2	0 - 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			
OVA	NOT DETECTED		
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 FOR STOOL SAMPLE.		
ABO GROUP & RH TYPE, EDTA WHOLE BLOOD			
ABO GROUP	TYPE B		









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Test Report Status Results Biological Reference Interval Units <u>Final</u> 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 NOT SIGNIFICANT RELEVANT PAST HISTORY NOT SIGNIFICANT RELEVANT PERSONAL HISTORY MARRIED / VEG. DIET / NO ALLERGIES / NO SMOKING / NO ALCOHOL. MENSTRUAL HISTORY (FOR FEMALES) REGULAR :-35-4 LMP (FOR FEMALES) 25/03/2022 **RELEVANT FAMILY HISTORY** NOT SIGNIFICANT HISTORY OF MEDICATIONS NOT SIGNIFICANT **ANTHROPOMETRIC DATA & BMI** HEIGHT IN METERS 1.56 mts WEIGHT IN KGS. 66 Kgs BMI & Weight Status as follows: kg/sqmts BMI 27 Below 18.5: Underweight 18.5 - 24.9: Normal 25.0 - 29.9: Overweight 30.0 and Above: Obese **GENERAL EXAMINATION** MENTAL / EMOTIONAL STATE NORMAL PHYSICAL ATTITUDE NORMAL **GENERAL APPEARANCE / NUTRITIONAL STATUS** HEALTHY **BUILT / SKELETAL FRAMEWORK** AVERAGE FACIAL APPEARANCE NORMAL

NORMAL

NORMAL

NORMAL NORMAI

NORMAL

NOT ENLARGED

NOT ENLARGED OR TENDER



THYROID GLAND

CAROTID PULSATION

SKIN

NECK

UPPER LIMB

LOWER LIMB

NECK LYMPHATICS / SALIVARY GLANDS





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	NORMAL	
BREAST (FOR FEMALES)	NORMAL	
PULSE	BRUIT	ALL PERIPHERAL PULSES WELL FELT, NO CAROTID
RESPIRATORY RATE	NORMAL	
CARDIOVASCULAR SYSTEM		
BP	110/70 MM HG	mm/Hg
PERICARDIUM	(SUPINE) NORMAL	
APEX BEAT		
	NORMAL	
	NORMAL	
	ABSENT	
	NORMAL	
SIZE AND SHAPE OF CHEST	NORMAL	
MOVEMENTS OF CHEST	SYMMETRICAL	
BREATH SOUNDS INTENSITY	NORMAL	
BREATH SOUNDS QUALITY	VESICULAR (NORM	AL)
ADDED SOUNDS	ABSENT	
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		







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CONJUNCTIVA	NORMAL		
EYELIDS	NORMAL		
EYE MOVEMENTS	NORMAL		
CORNEA	NORMAL		
DISTANT VISION RIGHT EYE WITHOUT GLASSES	WITHIN NORMAL LIMIT		
DISTANT VISION LEFT EYE WITHOUT GLASSES	WITHIN NORMAL LIMIT		
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	NOT SIGNIFICANT		
RELEVANT LAB INVESTIGATIONS	WITHIN NORMAL LIMITS		
RELEVANT NON PATHOLOGY DIAGNOSTICS	ECG:- NORMAL		
	X-RAY:- NORMAL.		
REMARKS / RECOMMENDATIONS	USG:- NORMAL. NORMAL		

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

(<15) In patients with microcytic anaemia. This needs to be interpreted in line with chincar correlation and suspicion. Estimation of NDA2 remains the gold standard for diagnosing a case of beta thalassaemia trait. 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 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. 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 and when there are abnormalities of the red cells such as pointicytosis, spherocytosis or sickle cells.

Reference :

1. Nathan and Oski's Haematology of Infancy and Childhood, 5th edition

Paediatric reference intervals. AACC Press, 7th edition. Edited by S. Soldin
 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



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CLIENT'S NAME AND ADDRESS :

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Test Report Status <u>Final</u>	Results	Biological Reference Interval Units
REFERRING DOCTOR : SELF		CLIENT PATIENT ID :
DRAWN :	RECEIVED : 14/04/2022 11:08	REPORTED : 15/04/2022 14:58
ACCESSION NO : 0181VD000919	AGE : 30 Years SEX : Female	
PATIENT NAME : NEHA GUPTA		PATIENT ID : NEHAF091291181

SRI 1td

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 HbSC, HbCC, and HbSC and HbC 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

"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, 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. GLUCÓSE, 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.

CORONARY 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 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.

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.

Recommendations:

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







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hepatocellular injury, to determine liver health.AST levels increase during acute hepatitis, sometimes due to a viral infection, is chemia to the liver, chronic hepatitis.obstruction of bile ducts.cirrhosis.

Results

Biological Reference Interval

Units

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, 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. SERUM BLOOD UREA NITROGEN-

Causes of Increased levels

Test Report Status

Pre renal

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

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)

<u>Final</u>

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 Sclerosis

Nutritional tips to manage increased Uric acid levels

Drink plenty of fluidsLimit 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 alobulin

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







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CDI 1+d

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), 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 failure, metabolic 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 prolonged vomiting,

URINALYSIS-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-

Trilodo FANLE, SECOND FANLE, SECOND AND A STREAM AND A ST 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. for Total T4, TSH & Total T3

Below mentioned	are the guidelines f	or Pregnancy related	reference ranges for 1 of	tal
Levels in	TOTAL T4	TSH3G	TOTAL T3	
Pregnancy	(µg/dL)	(µIU/mL)	(ng/dL)	
First Trimester	6.6 - 12.4	0.1 - 2.5	81 - 190	
2nd Trimester	6.6 - 15.5	0.2 - 3.0	100 - 260	
3rd Trimester	6.6 - 15.5	0.3 - 3.0	100 - 260	
Below mentioned	are the guidelines f	or age related refere	nce ranges for T3 and T4	ŧ.
Т3		T4		
(ng/dL)		(up/dL)		

(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 on the report under biological reference range.

Reference:

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

STOOL: OVA & PARASITE-

Acute infective diarrhoea and gastroenteritis (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 if 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 amoebiasis 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 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.







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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.







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Units

MEDI WHEEL FULL BODY HEALTH CHECKUP BELOW 40FEMALE

ULTRASOUND ABDOMEN ULTRASOUND ABDOMEN NO ABNORMALITIES DETECTED

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

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Dr. Ushma Wartikar **Consultant Pathologist**



