



MANISH PATIL

Test Report Status

101, TILALE APT ROAD NO 1, GOREGAON WEST

Final

SRL Ltd
PRIME SQUARE BUILDING,PLOT NO 1,GAIWADI INDUSTRIAL
ESTATE,S.V. ROAD,GOREGAON (W)
Mumbai, 400062
MAHARASHTRA, INDIA

Biological Reference Interval Units

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

1.0 - 6.0

PATIENT NAME: MANISH PATIL PATIENT ID: MANIMO5019129

ACCESSION NO: 0002VE039842 AGE: 31 Years SEX: Male ABHA NO:

DRAWN: 19/05/2022 08:35 RECEIVED: 19/05/2022 08:37 REPORTED: 20/05/2022 12:44

Results

REFERRING DOCTOR: SELF CLIENT PATIENT ID:

MEDI WHEEL FULL BODY HEALTH CHECK UP B	ELOW 40 MALE			
BLOOD COUNTS,EDTA WHOLE BLOOD				
HEMOGLOBIN	14.6		13.0 - 17.0	g/dL
METHOD: PHOTOMETRIC MEASUREMENT	1410		15.0 17.0	g/ u.c
RED BLOOD CELL COUNT	5.34		4.5 - 5.5	mi l /μL
METHOD : COULTER PRINCIPLE				, [
WHITE BLOOD CELL COUNT	9.10		4.0 - 10.0	thou/µL
METHOD : COULTER PRINCIPLE				, ,
PLATELET COUNT	282		150 - 410	thou/µL
METHOD: ELECTRONIC IMPEDENCE & MICROSCOPY				
RBC AND PLATELET INDICES				
HEMATOCRIT	44.1		40.0 - 50.0	%
METHOD: CALCULATED PARAMETER				
MEAN CORPUSCULAR VOL	82.7	Low	83.0 - 101.0	fL
METHOD : DERIVED PARAMETER FROM RBC HISTOGRAM				
MEAN CORPUSCULAR HGB.	27.4		27.0 - 32.0	pg
METHOD: CALCULATED PARAMETER				
MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION METHOD: CALCULATED PARAMETER	33.2		31.5 - 34.5	g/dL
MENTZER INDEX	15.5			
RED CELL DISTRIBUTION WIDTH	14.2	High	11.6 - 14.0	%
METHOD : DERIVED PARAMETER FROM RBC HISTOGRAM			1110 1110	,,
MEAN PLATELET VOLUME	7.4		6.8 - 10.9	fL
METHOD : DERIVED PARAMETER FROM PLATELET HISTOGRAM				
WBC DIFFERENTIAL COUNT - NLR				
SEGMENTED NEUTROPHILS	61		40 - 80	%
METHOD: VCSN TECHNOLOGY/ MICROSCOPY				
ABSOLUTE NEUTROPHIL COUNT	5.55		2.0 - 7.0	thou/µL
METHOD: CALCULATED PARAMETER				
LYMPHOCYTES	25		20 - 40	%
METHOD: VCSN TECHNOLOGY/ MICROSCOPY				
ABSOLUTE LYMPHOCYTE COUNT	2.28		1.0 - 3.0	thou/µL
METHOD: CALCULATED PARAMETER				
NEUTROPHIL LYMPHOCYTE RATIO (NLR)	2.4			



METHOD : CALCULATED EOSINOPHILS



%





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METHOD: VCSN TECHNOLO					
ABSOLUTE EOSINOPHI		0.36		0.02 - 0.50	thou/µL
METHOD : CALCULATED PAR	AMETER				
MONOCYTES		10		2.0 - 10.0	%
METHOD: VCSN TECHNOLO					
ABSOLUTE MONOCYTE	COUNT	0.91		0.2 - 1.0	thou/µL
METHOD : CALCULATED PAR	AMETER				
BASOPHILS		0		0 - 1	%
METHOD: VCSN TECHNOLO	GY/ MICROSCOPY				
ABSOLUTE BASOPHIL (COUNT	0.00	Low	0.02 - 0.10	thou/µL
METHOD : CALCULATED PAR	AMETER				
* ERYTHRO SEDIMEN	NTATION RATE, BLOOD				
SEDIMENTATION RATE	(ESR)	7		0 - 14	mm at 1 hr
METHOD : AUTOMATED (PHO	OTOMETRICAL CAPILLARY STOPPED F	LOW KINETIC ANALYSIS)			
GLUCOSE, FASTING,	PLASMA				
GLUCOSE, FASTING, PI	LASMA	95		74 - 99	mg/dL
METHOD : SPECTROPHOTOM	METRY HEXOKINASE				
GLYCOSYLATED HEM	OGLOBIN, EDTA WHOLE	BLOOD			
GLYCOSYLATED HEMOG	GLOBIN (HBA1C)	5.6		Non-diabetic: < 5.7 Pre-diabetics: 5.7 - 6.4 Diabetics: > or = 6.5 ADA Target: 7.0 Action suggested: > 8.0	%
METHOD: ION-EXCHANGE	HPLC				
MEAN PLASMA GLUCOS METHOD: CALCULATED PAR		114.0		< 116.0	mg/dL
GLUCOSE, POST-PRA	NDIAL, PLASMA				
GLUCOSE, POST-PRANI	DIAL, PLASMA	100		70 - 139	mg/dL
METHOD: SPECTROPHOTOM	METRY HEXOKINASE				
CORONARY RISK PRO	OFILE (LIPID PROFILE),	SERUM			
CHOLESTEROL		208	High	Desirable cholesterol level < 200 Borderline high cholesterol 200 - 239 High cholesterol > / = 240	mg/dL

METHOD: SPECTROPHOTOMETRY, ENZYMATIC COLORIMETRIC - CHOLETSEROL OXIDASE, ESTERASE, PEROXIDASE







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TRIGLYCERIDES	223	High	Normal: < 150 Borderline high: 150 - 199 High: 200 - 499 Very High: >/= 500	mg/dL
METHOD : SPECTROPHOTOMETRY, ENZYMATIC ENDPOINT V	VITH GLYCEROL BLANK			
HDL CHOLESTEROL	39	Low	Low HDL cholesterol < 40 High HDL cholesterol > / = 60	mg/dL
METHOD: SPECTROPHOTOMETRY, HOMOGENEOUS DIRECT	ENZYMATIC COLORIMETRIC			
DIRECT LDL CHOLESTEROL	132	High	Optimal: < 100 Near optimal/above optimal 129 Borderline high: 130 - 159 High: 160 - 189 Very high: > / = 190	mg/dL : 100 -
METHOD : SPECTROPHOTOMETRY, HOMOGENEOUS ENZYMA	ATIC COLORIMETRIC			
NON HDL CHOLESTEROL	169	High	Desirable : < 130 Above Desirable : 130 -159 Borderline High : 160 - 189 High : 190 - 219 Very high : > / = 220	mg/dL
METHOD: CALCULATED PARAMETER				
CHOL/HDL RATIO METHOD: CALCULATED PARAMETER	5.3	High	Low Risk: 3,3 - 4,4 Average Risk: 4.5 - 7.0 Moderate Risk: 7,1 - 11,0 High Risk: > 11.0	
	2.4	Uiah	Desirable // avv Biolo v O.F. 3	0
LDL/HDL RATIO	3.4	nigii	Desirable/Low Risk: 0.5 - 3. Borderline/Moderate Risk: 3 6.0 High Risk: > 6.0	
METHOD : CALCULATED PARAMETER				
VERY LOW DENSITY LIPOPROTEIN	45.0	High	< or = 30.0	mg/dL
METHOD: CALCULATED PARAMETER				
LIVER FUNCTION PROFILE, SERUM				
BILIRUBIN, TOTAL METHOD: SPECTROPHOTOMETRY, COLORIMETRIC -DIAZO	1.81	High	Upto 1.2	mg/dL
BILIRUBIN, DIRECT	0.51	High	0.0 - 0.2	mg/dL
METHOD: SPECTROPHOTOMETRY, JENDRASSIK & GROFF -		911	010 012	mg/uL
BILIRUBIN, INDIRECT	1.30	High	0.1 - 1.0	mg/dL
METHOD: CALCULATED PARAMETER		9		1119/ 42
TOTAL PROTEIN	7.2		6.0 - 8.0	g/dL









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METHOD · SPECTROPHOTO	METRY, COLORIMETRIC -BIURET, RE	FAGENT BLANK SERUM BLAN	IK		
ALBUMIN	HEIRT, COLORINE INC. BIORET, RE	4.8	•••	3.97 - 4.94	g/dL
	METRY, BROMOCRESOL GREEN(BCG			3137 1131	9, 42
GLOBULIN		2.4		2.0 - 3.5	g/dL
METHOD : CALCULATED PA	RAMETER				5/ ~-
ALBUMIN/GLOBULIN F		2.0		1.0 - 2.1	RATIO
METHOD : CALCULATED PA					
	ANSFERASE (AST/SGOT)	24		Upto 40	U/L
	METRY, WITHOUT PYRIDOXAL PHOS	PHATE ACTIVATION(P5P) - I	FCC		-,-
ALANINE AMINOTRANS		34		Upto 41	U/L
	METRY, WITHOUT PYRIDOXAL PHOS	PHATE ACTIVATION(P5P) - I	FCC	1	,
ALKALINE PHOSPHATA	SE	98		40 - 129	U/L
METHOD : SPECTROPHOTO	METRY, PNPP, AMP BUFFER - IFCC				,
GAMMA GLUTAMYL TR	ANSFERASE (GGT)	33		< 60	U/L
METHOD : SPECTROPHOTO	METRY, ENZYMATIC COLORIMETRIC	- G-GLUTAMYL-CARBOXY-NI	TROANILIDE -	IFCC	,
LACTATE DEHYDROGE	NASE	170		< 232	U/L
METHOD : SPECTROPHOTO	METRY, LACTATE TO PYRUVATE - UV	-IFCC			
SERUM BLOOD UREA	NITROGEN				
BLOOD UREA NITROG	EN	6		6 - 20	mg/dL
METHOD : SPECTROPHOTO	METRY, UREASE -COLORIMETRIC				J
CREATININE, SERUM	4				
CREATININE		0.86	Low	0.90 - 1.30	mg/dL
METHOD : SPECTROPHOTO	METRY, JAFFE'S ALKALINE PICRATE I	KINETIC - RATE BLANKED - I	FCC-IDMS STA	NDARIZED	5, -
BUN/CREAT RATIO					
BUN/CREAT RATIO		6.98	Low	8 - 15	
METHOD : CALCULATED PA	RAMETER			-	
URIC ACID, SERUM					
URIC ACID		7,3	Hiah	3.4 - 7.0	mg/dL
	METRY, ENZYMATIC COLORIMETRIC-			J. 710	9, a=
TOTAL PROTEIN, SE					
TOTAL PROTEIN		7.2		6.0 - 8.0	g/dL
	METRY, COLORIMETRIC -BIURET, RE		IK	010 010	g/ u.c
ALBUMIN, SERUM	,				
ALBUMIN		4.8		3.97 - 4.94	g/dL
	METRY, BROMOCRESOL GREEN(BCG			J.J/ - T.JT	y/ uL
GLOBULIN	TETAT, DROPOCKESOE GREEN(BCG	, DIE DINDING			
GLOBULIN		2.4		2.0 - 3.5	a/di
GLODULIN		۷.4		2.0 - 3.3	g/dL









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METHOD : CALCULATED PAR					
ELECTROLYTES (NA/	K/CL), SERUM			100 145	10
SODIUM		141		136 - 145	mmo l /L
METHOD : ISE INDIRECT POTASSIUM		4.70		3,5 - 5,1	mmo l /L
METHOD : ISE INDIRECT		4.70		5,5 - 5,1	IIIIIOI/ L
CHLORIDE		105		98 - 106	mmo l /L
METHOD : ISE INDIRECT					
PHYSICAL EXAMINA	TION, URINE				
COLOR		PALE YELLOW			
APPEARANCE		CLEAR			
SPECIFIC GRAVITY		1,000	Low	1,010 - 1,030	
CHEMICAL EXAMINA	TION, URINE				
PH		6.5		5.00 - 7.50	
PROTEIN		NOT-DETECTED		NOT DETECTED	
GLUCOSE		NOT-DETECTED		NOT DETECTED	
KETONES		NOT-DETECTED		NOT DETECTED	
BLOOD		NOT-DETECTED		NOT DETECTED	
BILIRUBIN		NOT-DETECTED		NOT DETECTED	
UROBILINOGEN		NOT-DETECTED		NORMAL	
NITRITE		NOT-DETECTED		NOT DETECTED	
LEUKOCYTE ESTERASE		NOT-DETECTED		NOT DETECTED	
MICROSCOPIC EXAM	IINATION, URINE				
PUS CELL (WBC'S)		1-2		0-5	/HPF
EPITHELIAL CELLS		0-1			/HPF
ERYTHROCYTES (RBC'S	5)	NOT DETECTED		0-5	/HPF
CASTS		NOT DETECTED			
CRYSTALS		NOT DETECTED			
BACTERIA		NOT DETECTED		NOT DETECTED	
YEAST		NOT DTECTED		NOT DETECTED	
METHOD: URINE ROUTINE & MICROSCOPY EXAMINATION BY INTEGRATED AUTOMATED SYSTEM					

Comments

NOTE: KINDLY EXERT CAUTION DURING INTERPRETATION OF FINDINGS REPORTED IN URINALYSIS WHERE IN THE SAMPLE IS MORE THAN TWO HOURS OLD.







Cert. No. MC-2010

CLIENT CODE: C000138356 **CLIENT'S NAME AND ADDRESS:**

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Test Report Status	<u>Final</u>	Results	Biological Reference	Interval Units	
THYROID PANEL, SE	RUM				
T3		128.0	80.0 - 200.0	ng/dL	
METHOD : COMPETITIVE EL	ECTROCHEMILUMINESCEN	CE IMMUNOASSAY			
T4		7.67	5.10 - 14.10	μg/dL	
METHOD: COMPETITIVE ELECTROCHEMILUMINESCENCE IMMUNOASSAY					
TSH 3RD GENERATION	J	2.290	0.270 - 4.200	μIU/mL	
METHOD : SANDWICH ELEC	TROCHEMILUMINESCENCE	IMMUNOASSAY			

STOOL: OVA & PARASITE

TEST CANCELLED AS SPECIMEN NOT RECEIVED REMARK

ABO GROUP & RH TYPE, EDTA WHOLE BLOOD

ABO GROUP 0

METHOD: HAEMAGGLUTINATION (AUTOMATED)

RH TYPE **POSITIVE**

METHOD: HAEMAGGLUTINATION (AUTOMATED)

* XRAY-CHEST

IMPRESSION NO ABNORMALITY DETECTED

TMT OR ECHO

TMT OR ECHO **PENDING**

* ECG

ECG LAD

* MEDICAL HISTORY

RELEVANT PRESENT HISTORY FULLY VACCINATED FOR COVID 19

RELEVANT PAST HISTORY MINOR FRACTURE SINCE RIGHT LEG CHILDHOOD

COVID INFECTION MARCH 2021

RELEVANT PERSONAL HISTORY NOT SIGNIFICANT

RELEVANT FAMILY HISTORY HYPERTENSION - FATHER AND MOTHER

HISTORY OF MEDICATIONS **NOT SIGNIFICANT**

* ANTHROPOMETRIC DATA & BMI

HEIGHT IN METERS 1.74 mts WEIGHT IN KGS. 81 Kgs BMI 27 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**



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PHYSICAL ATTITUDE NORMAL

GENERAL APPEARANCE / NUTRITIONAL STATUS OVERWEIGHT

BUILT / SKELETAL FRAMEWORK AVERAGE

FACIAL APPEARANCE ASYMMETRICAL

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 72/MIN REGULAR, ALL PERIPHERAL PULSES WELL FELT, NO CAROTID

BRUIT

RESPIRATORY RATE NORMAL

* CARDIOVASCULAR SYSTEM

BP 120/80 MM HG mm/Hg

(SUPINE) NORMAL

PERICARDIUM NORMAL APEX BEAT NORMAL

HEART SOUNDS S1, S2 HEARD NORMALLY

MURMURS ABSENT

* RESPIRATORY SYSTEM

SIZE AND SHAPE OF CHEST NORMAL

MOVEMENTS OF CHEST SYMMETRICAL

BREATH SOUNDS INTENSITY NORMAL

BREATH SOUNDS QUALITY VESICULAR (NORMAL)

ADDED SOUNDS ABSENT

* PER ABDOMEN

APPEARANCE NORMAL VENOUS PROMINENCE ABSENT

LIVER NOT PALPABLE
SPLEEN NOT PALPABLE
HERNIA ABSENT

* CENTRAL NERVOUS SYSTEM



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HIGHER FUNCTIONS	NORMAL					
CRANIAL NERVES	NORMAL					
CEREBELLAR FUNCTIONS	NORMAL					
SENSORY SYSTEM	NORMAL					
MOTOR SYSTEM	NORMAL					
REFLEXES	NORMAL					
* MUSCULOSKELETAL SYSTEM						
SPINE	NORMAL					
JOINTS	NORMAL					
* BASIC EYE EXAMINATION						
CONJUNCTIVA	NORMAL					
EYELIDS	NORMAL					
EYE MOVEMENTS	NORMAL					
CORNEA	NORMAL					
DISTANT VISION RIGHT EYE WITHOUT GLAS	SES WITHIN NORMAL LI	MIT (6/6)				
DISTANT VISION LEFT EYE WITHOUT GLASSE	S WITHIN NORMAL L	MIT (6/6)				
NEAR VISION RIGHT EYE WITHOUT GLASSES	WITHIN NORMAL LI	MIT (N6)				
NEAR VISION LEFT EYE WITHOUT GLASSES	WITHIN NORMAL L	MIT (N6)				
COLOUR VISION	NORMAL (17/17)					
* BASIC ENT EXAMINATION						
EXTERNAL EAR CANAL	NORMAL					
TYMPANIC MEMBRANE	NORMAL					
NOSE	NO ABNORMALITY (DETECTED				
SINUSES	NORMAL					
THROAT	NO ABNORMALITY (DETECTED				
TONSILS	NOT ENLARGED					
* BASIC DENTAL EXAMINATION						
TEETH	NORMAL					
GUMS	HEALTHY					
* SUMMARY						
RELEVANT HISTORY	NOT SIGNIFICANT					

RELEVANT HISTORY NOT SIGNIFICANT
RELEVANT GP EXAMINATION FINDINGS NOT SIGNIFICANT









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RAISED CHOLESTEROL (208) RELEVANT LAB INVESTIGATIONS RAISED TRIGLYCERIDES (223)

LOW HDL CHOLESTEROL (39) RAISED DIRECT LDL (132) RAISED NON HDL (169) RAISED VLDL (45)

RAISED BILIRÙBIN TOTAL (1.81) RAISED DIRECT BILIRUBIN (0.51) RAISED INDIRECT BILIRUBIN (1.30)

RAISED URIC ACID (7.3) USG-GRADE I FATTY LIVER

RELEVANT NON PATHOLOGY DIAGNOSTICS

REMARKS / RECOMMENDATIONS DYSLIPIDEMIA, RAISED BILIRUBIN, RAISED URIC ACID

REDUCE PROCESSED FOODS IN DIET

PURINE RICH FOODS MONITOR URIC ACID

FOLLOW UP WITH PHYSICIAN FOR RAISED LIPID PROFILE, RAISED URIC

ACID

Interpretation(s)

BLOOD COUNTS, EDTA 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. 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 - 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-

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







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ACCESSION NO: 0002VE039842 AGE: 31 Years SEX: Male ABHA NO:

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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, 879-884.
- 2. Forsham PH. Diabetes Mellitus: A rational plan for management. Postgrad Med 1982, 71,139-154.
- 3. Mayer TK, Freedman ZR: Protein glycosylation in Diabetes Mellitus: A review of laboratory measurements and their clinical utility. Clin Chim Acta 1983, 127, 147-184. GLUCOSE, 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

CORONARY RISK PROFILE (LIPID PROFILE), SERUMSerum 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 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, spieen, 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







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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

Post Renal

Malignancy, Nephrolithiasis, Prostatism

Causes of decreased levels

- Liver diseaseSIADH.

CREATININE, SERUM-

Higher than normal level may be due to:
• Blockage in the urinary tract

- Kidney problems, such as kidney damage or failure, infection, or reduced blood flow
- Loss of body fluid (dehydration)
- Muscle problems, such as breakdown of muscle fibers
- Problems during pregnancy, such as seizures (eclampsia)), or high blood pressure caused by pregnancy (preeclampsia)

Lower than normal level may be due to:

- Myasthenia Gravis
- Muscular dystrophy URIC ACID, SERUM-

Causes of Increased levels

- Dietary

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

Lesch nyhan syndrome. Type 2 DM.

Metabolic syndrome.

Causes of decreased levels

- Low Zinc Intake
- OCP's
- Multiple Sclerosis

Nutritional tips to manage increased Uric acid levels

- Drink plenty of fluidsLimit 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

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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







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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,

THYROID PANEL, SERUMTriiodothyronine T3 , is a thyroid hormone. It affects almost every physiological process in the body, including growth, development, metabolism, body temperature, and heart rate. Production of T3 and its prohormone thyroxine (T4) is activated by thyroid-stimulating hormone (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

TOTAL T4 TOTAL T3 Levels in TSH3Ġ (μIU/mL) 0.1 - 2.5 0.2 - 3.0 0.3 - 3.0 Pregnancy First Trimester (µg/dL) 6.6 - 12.4 (ng/dL) 81 - 190 100 - 260 100 - 260 2nd Trimester 6.6 - 15.5 6.6 - 15.5 3rd Trimester

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

(µg/dL) (ng/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.

- 1. Burtis C.A., Ashwood E. R. Bruns D.E. Teitz textbook of Clinical Chemistry and Molecular Diagnostics, 4th Edition.
 2. Gowenlock A.H. Varley's Practical Clinical Biochemistry, 6th Edition.
 3. 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.

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.

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MEDI WHEEL FULL BODY HEALTH CHECK UP BELOW 40 MALE

* ULTRASOUND ABDOMEN **ULTRASOUND ABDOMEN** GRADE I FATTY LIVER.

End Of Report

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