





CLIENT'S NAME AND ADDRESS : ACROFEMI HEALTHCARE LTD (MEDIWHEEL) F-703, LADO SARAI, MEHRAULI SOUTH WEST DELHI NEW DELHI 110030 DELHI INDIA 8800465156

SRL Ltd Shop CG 017, PALM SPRINGS PLAZA GURUGRAM, 122001 HARYANA, INDIA Tel : 9111591115

PATIENT NAME : SUNIL KUMAR		PATIENT ID : SUNIM140190282
ACCESSION NO : 0282VH000525	AGE : 32 Years SEX : Male	ABHA NO :
DRAWN :	RECEIVED : 09/08/2022 12:36	REPORTED : 10/08/2022 07:44
REFERRING DOCTOR : SELF		CLIENT PATIENT ID :

Test Report Status Final Results Biological Reference Interval	l Units
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MEDI WHEEL FULL BODY HEALTH CHECK UP BELOW 40 MALE

BLOOD COUNTS,EDTA WHOLE BLOOD				
HEMOGLOBIN	15.8		13.0 - 17.0	g/dL
METHOD : SPECTROPHOTOMETRY				5,
RED BLOOD CELL COUNT	5.29		4.5 - 5.5	mil/µL
METHOD : IMPEDANCE				
WHITE BLOOD CELL COUNT	5.70		4.0 - 10.0	thou/µL
METHOD : IMPEDANCE				
PLATELET COUNT	182		150 - 410	thou/µL
METHOD : IMPEDANCE				
RBC AND PLATELET INDICES				
HEMATOCRIT	49.2		40 - 50	%
METHOD : CALCULATED				
MEAN CORPUSCULAR VOL	93.0		83 - 101	fL
METHOD : DERIVED FROM IMPEDANCE MEASURE				
MEAN CORPUSCULAR HGB.	29.8		27.0 - 32.0	pg
METHOD : CALCULATED PARAMETER				
MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION METHOD : CALCULATED PARAMETER	32.0		31.5 - 34.5	g/dL
MENTZER INDEX	17.6			
RED CELL DISTRIBUTION WIDTH	14.8	High	11.6 - 14.0	%
METHOD : DERIVED FROM IMPEDANCE MEASURE				
MEAN PLATELET VOLUME	12.9	High	6.8 - 10.9	fL
METHOD : DERIVED FROM IMPEDANCE MEASURE				
WBC DIFFERENTIAL COUNT - NLR				
SEGMENTED NEUTROPHILS	44		40 - 80	%
METHOD : DHSS FLOWCYTOMETRY				
ABSOLUTE NEUTROPHIL COUNT	2.50		2.0 - 7.0	thou/µL
METHOD : DHSS FLOWCYTOMETRY, CALCULATED				
LYMPHOCYTES	48	High	20 - 40	%
METHOD : DHSS FLOWCYTOMETRY				
ABSOLUTE LYMPHOCYTE COUNT	2.75		1 - 3	thou/µL
METHOD : DHSS FLOWCYTOMETRY, CALCULATED				
NEUTROPHIL LYMPHOCYTE RATIO (NLR)	0.9			
METHOD : CALCULATED				
EOSINOPHILS	02		1 - 6	%











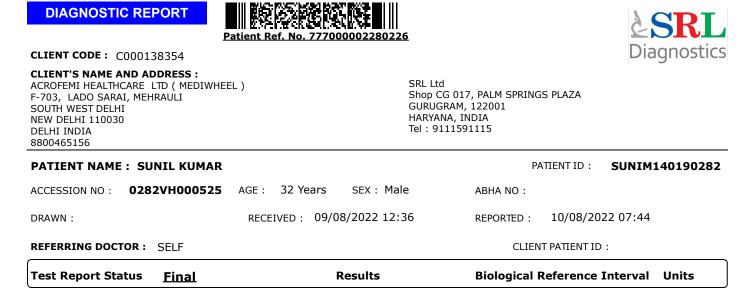
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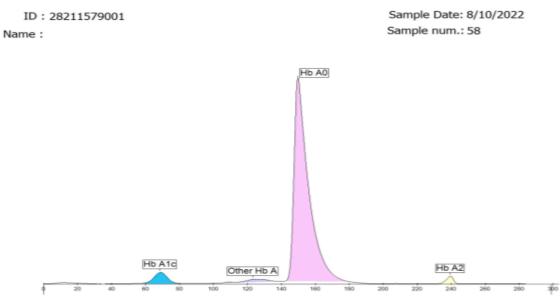
PATIENT NAME : SUNIL KUMAR		PATIENT ID : SUNIM14019028
ACCESSION NO : 0282VH000525	AGE : 32 Years SEX : Male	ABHA NO :
DRAWN :	RECEIVED : 09/08/2022 12:36	REPORTED : 10/08/2022 07:44
REFERRING DOCTOR : SELF		CLIENT PATIENT ID :
Test Report Status <u>Final</u>	Results	Biological Reference Interval Units
METHOD : DHSS FLOWCYTOMETRY		
ABSOLUTE EOSINOPHIL COUNT	0.11	0.02 - 0.50 thou/µL
METHOD : DHSS FLOWCYTOMETRY, CALCULATED		
MONOCYTES METHOD : DHSS FLOWCYTOMETRY	06	2 - 10 %
ABSOLUTE MONOCYTE COUNT	0.34	0.20 - 1.00 thou/µL
METHOD : DHSS FLOWCYTOMETRY, CALCULATED		
BASOPHILS	00	0 - 2 %
METHOD : IMPEDANCE		
ABSOLUTE BASOPHIL COUNT	00 Low	0.02 - 0.10 thou/µL
METHOD : DHSS FLOWCYTOMETRY, CALCULATED)	
ERYTHRO SEDIMENTATION RATE,	BLOOD	
SEDIMENTATION RATE (ESR)	2	0 - 14 mm at 1 hr
METHOD : AUTOMATED (PHOTOMETRICAL CAPIL	LARY STOPPED FLOW KINETIC ANALYSIS)	
GLUCOSE, FASTING, PLASMA		
GLUCOSE, FASTING, PLASMA	88	Normal 75 - 99 mg/dL Pre-diabetics: 100 - 125 Diabetic: > or = 126
METHOD : SPECTROPHOTOMETRY HEXOKINASE		
GLYCOSYLATED HEMOGLOBIN, ED		
GLYCOSYLATED HEMOGLOBIN (HBA1	C) 5.3	Non-diabetic: < 5.7 % Pre-diabetics: 5.7 - 6.4 Diabetics: > or = 6.5 ADA Target: 7.0 Action suggested: > 8.0
METHOD : CAPILLARY ELECTROPHORESIS		
MEAN PLASMA GLUCOSE	105.4	< 116 mg/dL
METHOD : CALCULATED PARAMETER		







PLOT NO.31, ELECTRONIC CITY, SECTOR 18, GURUGRAM



A1c Haemoglobin Electrophoresis

Fractions	%	mmol/mol	Cal. %	
Hb A1c	-	35	5.3	
Other Hb A	2.0			
Hb AO	91.5			
Hb A2	1.8			

70 - 139

HbA1c % cal :5.3 %

91

Comments :

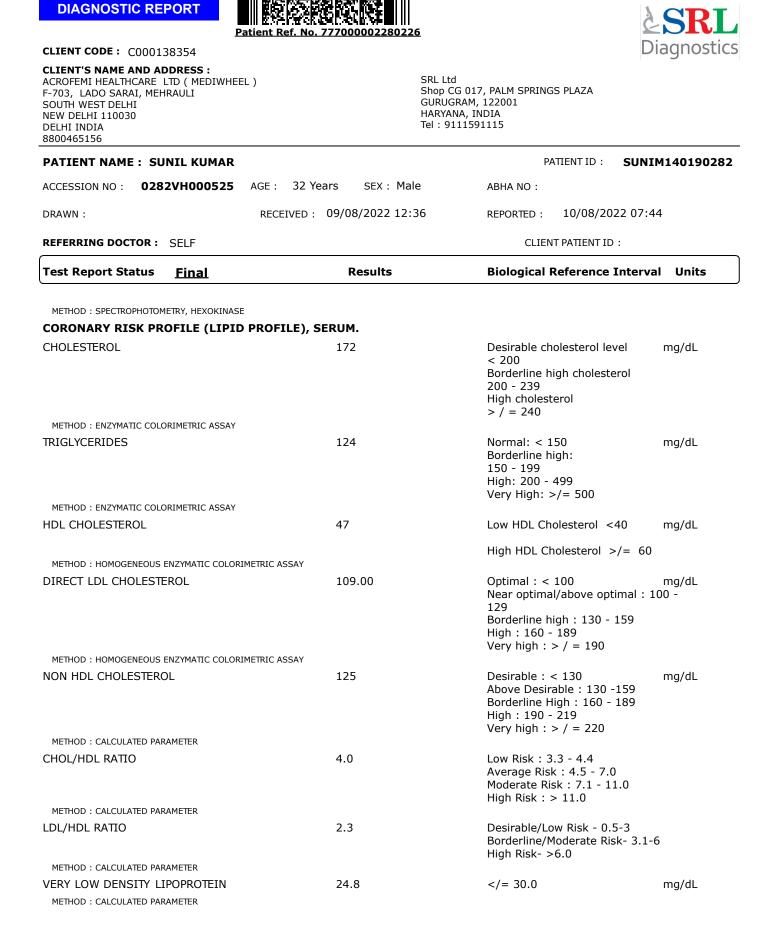
GLUCOSE, POST-PRANDIAL, PLASMA

GLUCOSE, POST-PRANDIAL, PLASMA

Scan to View Details

mg/dL















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Interpretation(s)

Serum lipid profile is measured for cardiovascular risk prediction, the test includes five basic parameters: total cholesterol, triglycerides, HDL cholesterol, LDL cholesterol and Non HDL cholesterol.

1) Cholesterol levels help assess the patient risk status and to follow the progress of patient under treatment to lower serum cholesterol concentrations.

2) Serum Triglyceride (TG) are a type of fat and a major source of energy for the body.

- Both quantity and composition of the diet impact on plasma triglyceride concentrations
- Elevations in TG levels are the result of overproduction and impaired clearance.
- High TG are associated with increased risk for CAD (Coronary artery disease) in patients with other risk factors, such as low HDL-C, some

patient groups with elevated apolipoprotein B concentrations, and patients with forms of LDL that may be particularly atherogenic. 3)HDL-C plays a crucial role in the initial step of reverse cholesterol transport, this considered to be the primary atheroprotective function of HDL

4) LDL -C plays a key role in causing and influencing the progression of atherosclerosis and, in particular, coronary sclerosis.

The majority of cholesterol stored in atherosclerotic plaques originates from LDL, thus LDL-C value is the most powerful clinical predictor. 5)Non HDL cholesterol: Non-HDL-C measures the cholesterol content of all atherogenic lipoproteins, including LDL hence it is a better marker of risk in both primary and secondary prevention studies.

Non-HDL-C also covers, to some extent, the excess ASCVD risk imparted by the sdLDL, which is significantly more atherogenic than the normal large buoyant particles, an elevated non-HDL-C indirectly suggests greater proportion of the small, dense variety of LDL particles Lipid Association of India recommends LDL-C as primary target and Non HDL-C as co-primary treatment target.

Risk Stratification for ASCVD (Atherosclerotic cardiovascular disease) by Lipid Association of India

	Risk Category		
Extreme risk group	A.CAD with > 1 feature of high risk group		
	B. CAD with > 1 feature of Very high risk group or recurrent ACS (within 1 year) despite LDL-C < or = 50 mg/dl or polyvascular disease		
Very High Risk	1. Established ASCVD 2. Diabetes with 2 major risk factors or evidence of end organ damage 3. Familial Homozygous Hypercholesterolemia		
High Risk	 Three major ASCVD risk factors. Diabetes with 1 major risk factor or no evidence of end organ damage. CKD stage 3B or 4. LDL >190 mg/dl Extreme of a single risk factor. Coronary Artery Calcium - CAC >300 AU. Lipoprotein a >/= 50mg/dl Non stenotic carotid plaque 		
Moderate Risk	2 major ASCVD risk factors		
Low Risk	0-1 major ASCVD risk factors		
Major ASCVD (Atherosclerotic cardiovascular disease) Risk Factors			
1. Age > or = 45 years in males and > or = 55 years in females 3. Current Cigarette smoking or tobacco use			
2. Family history of pre	2. Family history of premature ASCVD 4. High blood pressure		
5 Low HDL			











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Newer treatment goals and statin initiation thresholds based on the risk categories proposed by LAI in 2020

Risk Group	Treatment Goals		Consider Drug Therapy	
	LDL-C (mg/dl)	Non-HDL (mg/dl)	LDL-C (mg/dl)	Non-HDL (mg/dl)
Extreme Risk Group	<50 (Optional goal	< 80 (Optional goal	>OR = 50	>OR = 80
Category A	< OR = 30)	<or 60)<="" =="" td=""><td></td><td></td></or>		
Extreme Risk Group	<or 30<="" =="" td=""><td><or 60<="" =="" td=""><td>> 30</td><td>>60</td></or></td></or>	<or 60<="" =="" td=""><td>> 30</td><td>>60</td></or>	> 30	>60
Category B				
Very High Risk	<50	<80	>OR= 50	>OR= 80
High Risk	<70	<100	>OR= 70	>OR= 100
Moderate Risk	<100	<130	>OR= 100	>OR= 130
Low Risk	<100	<130	>OR= 130*	>OR= 160

*After an adequate non-pharmacological intervention for at least 3 months

References:

Management of Dyslipidaemia for the Prevention of Stroke: Clinical Practice Recommendations from the Lipid Association of India. Current Vascular Pharmacology, 2022, 20, 134-155.

LIVER FUNCTION PROFILE, SERUM				
BILIRUBIN, TOTAL	0.4		Upto 1.2	mg/dL
METHOD : COLORIMETRIC DIAZO METHOD				
BILIRUBIN, DIRECT	0.2		< 0.30	mg/dL
METHOD : COLORIMETRIC DIAZO METHOD				
BILIRUBIN, INDIRECT	0.20		0.1 - 1.0	mg/dL
METHOD : CALCULATED PARAMETER				
TOTAL PROTEIN	7.9		6.0 - 8.0	g/dL
METHOD : SPECTROPHOTOMETRY, BIURET				
ALBUMIN	5.1	High	3.97 - 4.94	g/dL
METHOD : SPECTROPHOTOMETRY, BROMOCRESOL GREEN(BCG)	- DYE BINDING			
GLOBULIN	2.9		2.0 - 3.5	g/dL
METHOD : CALCULATED PARAMETER				
ALBUMIN/GLOBULIN RATIO	1.8		1.0 - 2.1	RATIO
METHOD : CALCULATED PARAMETER				
ASPARTATE AMINOTRANSFERASE (AST/SGOT)	35		< OR = 50	U/L
METHOD : SPECTROPHOTOMETRY, WITH PYRIDOXAL PHOSPHATE	ACTIVATION-IFCC			
ALANINE AMINOTRANSFERASE (ALT/SGPT)	72	High	< OR = 50	U/L
METHOD : SPECTROPHOTOMETRY, WITH PYRIDOXAL PHOSPHATE	ACTIVATION-IFCC			
ALKALINE PHOSPHATASE	95		40 - 129	U/L
METHOD : SPECTROPHOTOMETRY, PNPP, AMP BUFFER - IFCC				
GAMMA GLUTAMYL TRANSFERASE (GGT)	29		0 - 60	U/L











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Test Report Status <u>Final</u>	Results		Biological Reference	Interval Units
			125 220	11/1
			125 - 220	U/L
METHOD : SPECTROPHOTOMETRY, LACTATE TO SERUM BLOOD UREA NITROGEN				
BLOOD UREA NITROGEN	7.0		6 - 20	ma/dl
	2.0 EST WITH UREASE AND GLUTAMATE DEHYDROGENASE		0 - 20	mg/dL
CREATININE, SERUM				
CREATININE	0.90		0.7 - 1.2	mg/dL
METHOD : SPECTROPHOTOMETRIC, JAFFE'S K			0.7 1.2	ing/dL
BUN/CREAT RATIO				
BUN/CREAT RATIO	7.78	Low	8.0 - 15.0	
METHOD : CALCULATED PARAMETER				
URIC ACID, SERUM				
URIC ACID	5.0		3.4 - 7.0	mg/dL
METHOD : SPECTROPHOTOMETRY, URICASE				
TOTAL PROTEIN, SERUM				
TOTAL PROTEIN	7.9		6.0 - 8.0	g/dL
METHOD : SPECTROPHOTOMETRY, BIURET				
ALBUMIN, SERUM				
ALBUMIN	5.1	High	3.97 - 4.94	g/dL
METHOD : SPECTROPHOTOMETRY, BROMOCRE	ESOL GREEN(BCG) - DYE BINDING			
GLOBULIN				
GLOBULIN	2.9		2.0 - 3.5	g/dL
METHOD : CALCULATED PARAMETER				
ELECTROLYTES (NA/K/CL), SER				
SODIUM	141		136 - 145	mmol/L
	4.5	Ll:ab		
	4.6	нıgn	3.5 - 4.5	mmol/L
METHOD : ISE INDIRECT CHLORIDE	105		98 - 107	mmol/L
METHOD : ISE INDIRECT	105		50 107	
PHYSICAL EXAMINATION, URIN	E			
COLOR	PALE YELLOW			
APPEARANCE	CLEAR			
SPECIFIC GRAVITY	<=1.005		1.003 - 1.035	
	~=1.005		1.005 1.055	











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<u>Final</u>

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Comments

NOTE : MICROSCOPIC EXAMINATION OF URINE IS PERFORMED ON CENTRIFUGED URINARY SEDIMENT IN NORMAL URINE SAMPLES CAST AND CRYSTALS ARE NOT DETECTED. CHEMICAL EXAMINATION, URINE PH 6.0 4.7 - 7.5 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 NORMAL NORMAL NITRITE NOT DETECTED NOT DETECTED LEUKOCYTE ESTERASE NOT DETECTED NOT DETECTED **MICROSCOPIC EXAMINATION, URINE** 0-1 PUS CELL (WBC'S) 0-5 EPITHELIAL CELLS 0-5 0 - 1ERYTHROCYTES (RBC'S) NOT DETECTED NOT DETECTED CASTS NOT DETECTED CRYSTALS NOT DETECTED BACTERIA NOT DETECTED NOT DETECTED **THYROID PANEL, SERUM** 131.0 80 - 200 T3 METHOD : ELECTROCHEMILUMINESCENCE IMMUNO ASSAY Τ4 8.30 5.1 - 14.1 METHOD : ELECTROCHEMILUMINESCENCE IMMUNO ASSAY 0.27 - 4.2 TSH 3RD GENERATION 2.650 METHOD : ELECTROCHEMILUMINESCENCE IMMUNO ASSAY **STOOL: OVA & PARASITE** COLOUR YELLOW CONSISTENCY SEMI FORMED ODOUR FOUL NOT DETECTED MUCUS ABSENT VISIBLE BLOOD ABSENT ABSENT 0 - 5 POLYMORPHONUCLEAR LEUKOCYTES NOT DETECTED





/HPF

/HPF

/HPF

/HPF

ng/dL

µg/dL

µIU/mL







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RED BLOOD CELLS	NOT DETECTED	NOT DETECTED /HPF
MACROPHAGES	NOT DETECTED	NOT DETECTED
CHARCOT-LEYDEN CRYSTALS	NOT DETECTED	NOT DETECTED
TROPHOZOITES	NOT DETECTED	NOT DETECTED
CYSTS	NOT DETECTED	NOT DETECTED
OVA	NOT DETECTED	
LARVAE	NOT DETECTED	NOT DETECTED
ADULT PARASITE	NOT DETECTED	
ABO GROUP & RH TYPE, EDTA WHOLE BLOOD		
ABO GROUP	0	
METHOD : HEMAGGLUTINATION REACTION ON SOLID PHASE		
RH TYPE	RH+	
METHOD : HEMAGGLUTINATION REACTION ON SOLID PHASE		
XRAY-CHEST		
»»	BOTH THE LUNG FIELDS A	
»»	BOTH THE COSTOPHRENI	C AND CARDIOPHRENIC ANGLES ARE CLEAR
»»	BOTH THE HILA ARE NOR	MAL
»»	CARDIAC AND AORTIC SH	IADOWS APPEAR NORMAL
»»	BOTH THE DOMES OF THE	DIAPHRAGM ARE NORMAL
»»	VISUALIZED BONY THORA	AX IS NORMAL
IMPRESSION	NO ABNORMALITY DETEC	TED
TMT OR ECHO		
TMT OR ECHO	ECHO DONE	
ECG	No RWMA Mild PR, Trivial Normal LV syst Normal LV dias Normal LV dias No Clot/Vegeta	cardiac chambers and normal valves MR tolic function LVEF ~ 60 % stolic function, E>A ation/Pericardial Effusion ,no flow seen across.
ECG	WITHIN NORMAL LIMITS	
MEDICAL HISTORY		
RELEVANT PRESENT HISTORY	H/O ACIDITY - 2 YRS	
RELEVANT PAST HISTORY	NOT SIGNIFICANT	
RELEVANT PERSONAL HISTORY	NON SMOKER NO ALCOH	OL
RELEVANT FAMILY HISTORY	NOT SIGNIFICANT	
	-	



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PATIENT NAME : SUNIL KUMAR		PATIENT ID : SUNIM140190
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Test Report Status <u>Final</u>	Results	Biological Reference Interval Units
OCCUPATIONAL HISTORY	SERVICE	
HISTORY OF MEDICATIONS	NOT SIGNIFICANT	
ANTHROPOMETRIC DATA & BMI		
HEIGHT IN METERS	1.71	mts
WEIGHT IN KGS.	69.1	Kgs
ВМІ	24	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		
1ENTAL / EMOTIONAL STATE	NORMAL	
PHYSICAL ATTITUDE	NORMAL	
GENERAL APPEARANCE / NUTRITIONAL STATUS	HEALTHY	
BUILT / SKELETAL FRAMEWORK	AVERAGE	
ACIAL APPEARANCE	NORMAL	
SKIN	NORMAL	
JPPER LIMB	NORMAL	
LOWER LIMB	NORMAL	
NECK	NORMAL	
NECK LYMPHATICS / SALIVARY GLANDS	NOT ENLARGED OR TEN	NDER
HYROID GLAND	NOT ENLARGED	
CAROTID PULSATION	NORMAL	
TEMPERATURE	NORMAL	
PULSE		RIPHERAL PULSES WELL FELT
RESPIRATORY RATE	NORMAL	
CARDIOVASCULAR SYSTEM		
3P	120/84 MMHG (SUPINE)	mm/Hg
PERICARDIUM	NORMAL	
APEX BEAT	NORMAL	
HEART SOUNDS	NORMAL	
MURMURS	ABSENT	
SIZE AND SHAPE OF CHEST	NORMAL	
MOVEMENTS OF CHEST	SYMMETRICAL	











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ACCESSION NO : 0282VH000525	AGE : 32 Years SEX : Male	ABHA NO :
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Test Report Status	inal	Results	Biological Reference Interval	Units
BREATH SOUNDS INTENS	SITY	NORMAL		
BREATH SOUNDS QUALIT	Υ	VESICULAR (NORMAL)		
ADDED SOUNDS		ABSENT		
PER ABDOMEN				
APPEARANCE		NORMAL		
VENOUS PROMINENCE		ABSENT		
LIVER		NOT PALPABLE		
SPLEEN		NOT PALPABLE		
CENTRAL NERVOUS SY	STEM			
HIGHER FUNCTIONS		NORMAL		
CRANIAL NERVES		NORMAL		
CEREBELLAR FUNCTIONS		NORMAL		
SENSORY SYSTEM		NORMAL		
MOTOR SYSTEM		NORMAL		
REFLEXES		NORMAL		
MUSCULOSKELETAL SY	STEM			
SPINE		NORMAL		
JOINTS		NORMAL		
BASIC EYE EXAMINATI	ON			
DISTANT VISION RIGHT	EYE WITHOUT GLASSES	6/6		
DISTANT VISION LEFT EY	E WITHOUT GLASSES	6/6		
NEAR VISION RIGHT EYE	WITHOUT GLASSES	N/6		
NEAR VISION LEFT EYE V	VITHOUT GLASSES	N/6		
COLOUR VISION		17/17		
SUMMARY				
REMARKS / RECOMMEND	ATIONS			
		ADVISED LIFESTYLE CHANGES		
		REVIEW WITH MD PHYSIC	CIAN WITH ALL REPORTS FOR FURTH	ER ADVICE

REVIEW WITH MD PHYSICIAN WITH ALL REPORTS FOR FURTHER ADVICE AND MANAGEMENT. ADVISED GASTEROENTEROLOGY CONSULTATION IN VIEW OF SYMPTOMS.

Interpretation(s)











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BLOOD COUNTS, EDTA WHOLE BLOOD-

The cell morphology is well preserved for 24hrs. However after 24-48 hrs a progressive increase in MCV and HCT is observed leading to a decrease in MCHC. A direct smear is recommended for an accurate differential count and for examination of RBC morphology.

RBC AND PLATELET INDICES-

Mentzer index (MCV/RBC) is an automated cell-counter based calculated screen tool to differentiate cases of Iron deficiency anaemia(>13) from Beta thalassaemia trait (<13) in patients with microcytic anaemia. This needs to be interpreted in line with clinical correlation and suspicion. Estimation of HbA2 remains the gold standard for

diagnosing a case of beta thalassaemia trait. WBC DIFFERENTIAL COUNT - 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 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 pointicotytosis, 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

GLYCOSYLATED HEMOGLOBIN, EDTA WHOLE BLOOD-

Glycosylated hemoglobin (GHb) has been firmly established as an index of long-term blood glucose concentrations and as a measure of the risk for the development of complications in patients with diabetes mellitus. Formation of GHb is essentially irreversible, and the concentration in the blood depends on both the life span of the red blood cell (average 120 days) and the blood glucose concentration. Because the rate of formation of GHb is directly proportional to the concentration of glucose in the blood, the GHb concentration represents the integrated values for glucose over the preceding 6-8 weeks.

Any condition that alters the life span of the red blood cells has the potential to alter the GHb level. Samples from patients with hemolytic anemias will exhibit decreased glycated hemoglobin values due to the shortened life span of the red cells. This effect will depend upon the severity of the anemia. Samples from patients with polycythemia or post-splenectomy may exhibit increased glycated hemoglobin values due to a somewhat longer life span of the red cells.

Glycosylated hemoglobins results from patients with HbSS, HbCC, and HbSC and 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 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.

 Sorsham PH. Diabetes Mellitus: A rational plan for management. Postgrad Med 1982, 71,139-154.
 Mayer TK, Freedman ZR: Protein glycosylation in Diabetes Mellitus: A review of laboratory measurements and their clinical utility. Clin Chim Acta 1983, 127, 147-184. GLUCOSE, POST-PRANDIAL, PLASMA-ADA Guidelines for 2hr post prandial glucose levels is only after ingestion of 75grams of glucose in 300 ml water, over a period of 5 minutes

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 beautifue of bile ducts circhesie. 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







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HARYANA, INDIA
Tel : 9111591115

Test Report Status <u>Final</u>	Results	Biological Reference Interval Units
REFERRING DOCTOR : SELF		CLIENT PATIENT ID :
DRAWN :	RECEIVED : 09/08/2022 12:36	REPORTED : 10/08/2022 07:44
ACCESSION NO : 0282VH000525	AGE : 32 Years SEX : Male	ABHA NO :
PATIENT NAME : SUNIL KUMAR		PATIENT ID : SUNIM140190282

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 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) 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 fluids
Limit animal proteins High Fibre foods
 Vit C Intake Antioxidant rich foods TOTAL PROTEIN, SERUM Serum total protein, also known as total protein, is a biochemical test for measuring the total amount of protein in serum. Protein in the plasma is made up of albumin and 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 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,











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Addison's disease, metabolic acidosis, acute starvation, dehydration, and with rapid K infusion. Chloride is increased in dehydration, renal tubular acidosis (hyperchloremia metabolic acidosis), acute renal failure, metabolic acidosis associated with prolonged diarrhea and loss of sodium bicarbonate, diabetes insipidus, adrenocortical hyperfuction, salicylate intoxication and with excessive infusion of isotonic saline or extremely high dietary intake of salt. Chloride is decreased in overhydration, chronic respiratory acidosis, salt-losing nephritis, metabolic alkalosis, congestive heart failure, Addisonian crisis, certain types of metabolic acidosis, persistent gastric secretion and prolonged vomiting,

MICROSCOPIC EXAMINATION, URINE-

Routine unalysis assists in screening and diagnosis of various metabolic, urological, kidney and liver disorders Protein: Elevated proteins can be an early sign of kidney disease. Urinary protein excretion can also be temporarily elevated by strenuous exercise, orthostatic proteinuria, dehydration, urinary tract infections and acute illness with fever Glucose: Uncontrolled diabetes mellitus can lead to presence of glucose in urine. Other causes include pregnancy, hormonal disturbances, liver disease and certain

medications.

Ketones: Uncontrolled diabetes mellitus can lead to presence of ketones in urine. Ketones can also be seen in starvation, frequent vomiting, pregnancy and strenuous exercise.

Blood: Occult blood can occur in urine as intact erythrocytes or haemoglobin, which can occur in various urological, nephrological and bleeding disorders. Leukocytes: An increase in leukocytes is an indication of inflammation in urinary tract or kidneys. Most common cause is bacterial urinary tract infection.

Nitrite: Many bacteria give positive results when their number is high. Nitrite concentration during infection increases with length of time the urine specimen is retained in

bladder prior to collection. pH: The kidneys play an important role in maintaining acid base balance of the body. Conditions of the body producing acidosis/ alkalosis or ingestion of certain type of food can affect the pH of urine.

Specific gravity: Specific gravity gives an indication of how concentrated the urine is. Increased specific gravity is seen in conditions like dehydration, glycosuria and proteinuria while decreased specific gravity is seen in excessive fluid intake, renal failure and diabetes insipidus.

Bilirubin: In certain liver diseases such as biliary obstruction or hepatitis, bilirubin gets excreted in urine. Urobilinogen: Positive results are seen in liver diseases like hepatitis and cirrhosis and in cases of hemolytic anemia

THYROID PANEL, SERUM-

Triiodothyronine T3 , is a thyroid hormone. It affects almost every physiological process in the body, including growth, development, metabolism, body temperature, and heart rate. Production of T3 and its prohormone thyroxine (T4) is activated by thyroid-stimulating hormone (TSH), which is released from the pituitary gland. Elevated concentrations of T3, and T4 in the blood inhibit the production of TSH.

Thyroxine T4, Thyroxine's principal function is to stimulate the metabolism of all cells and tissues in the body. Excessive secretion of thyroxine in the body is hyperthyroidism, and deficient secretion is called hypothyroidism. Most of the thyroid hormone in blood is bound to transport proteins. Only a very small fraction of the circulating hormone is free and biologically active.

In primary hypothyroidism, TSH levels are significantly elevated, while in secondary and tertiary hypothyroidism, TSH levels are low. Below mentioned are the guidelines for Pregnancy related reference ranges for Total T4, TSH & Total T3

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 a	re the guidelines	for age related refere	nce ranges for T3 and	T4.
T2		TA		

T3	T4	
(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:

1. 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.

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.









DIAGNOSTIC REPORT

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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.











Units

CLIENT CODE: C000138354

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		×

Results

Test Report Status <u>Final</u>

MEDI WHEEL FULL BODY HEALTH CHECK UP BELOW 40 MALE

ULTRASOUND ABDOMEN ULTRASOUND ABDOMEN GRADE I FATTY LIVER

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

Dr. Arpita Roy, MD Pathologist



Dr. Mamta Kumari Consultant Microbiologist



Sr.Microbiologist Microbiologist

Dalling

Dr. Deblina Naithani Consultant Physician

CONDITIONS OF LABORATORY TESTING & REPORTING

 It is presumed that the test sample belongs to the patient named or identified in the test requisition form.
 All tests are performed and reported as per the turnaround time stated in the SRL Directory of Services.
 Result delays could occur due to unforeseen circumstances such as non-availability of kits / equipment breakdown / natural calamities / technical downtime or any other unforeseen event.

- 4. A requested test might not be performed if:
 - i. Specimen received is insufficient or inappropriate
 - ii. Specimen quality is unsatisfactory
 - iii. Incorrect specimen type

iv. Discrepancy between identification on specimen container label and test requisition form

5. SRL confirms that all tests have been performed or assayed with highest quality standards, clinical safety & technical integrity.

6. Laboratory results should not be interpreted in isolation; it must be correlated with clinical information and be interpreted by registered medical practitioners only to determine final diagnosis.

Biological Reference Interval

7. Test results may vary based on time of collection, physiological condition of the patient, current medication or nutritional and dietary changes. Please consult your doctor or call us for any clarification.

- 8. Test results cannot be used for Medico legal purposes.
- 9. In case of queries please call customer care
- (91115 91115) within 48 hours of the report.

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