

ACROFEMI HEALTHCARE LTD (MEDIWHEEL)

F-703, LADO SARAI, MEHRAULI SOUTH WEST DELHI

NEW DELHI 110030 DELHI INDIA 8800465156

PLOT No. 88, ROAD No. 15, MIDC ESTATE, ANDHERI (EAST)

MUMBAI, 400093 MAHARASHTRA, INDIA

Tel: 09152729959/9111591115, Fax:

CIN - U74899PB1995PLC045956

PATIENT NAME: SUJIT BHALERAO PATIENT ID: SUJIM535031940

0065VE001554 AGE: 33 Years ACCESSION NO: SEX: Male

DRAWN: RECEIVED: 14/05/2022 07:42 REPORTED: 16/05/2022 14:11

REFERRING DOCTOR: SELF CLIENT PATIENT ID:

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

MEDI WHEEL FULL BODY HEALTH CHECK UP BELOW 40 MALE

PHYSICAL EXAMINAT:	TON,	URINE
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COLOR PALE YELLOW

APPEARANCE CLEAR

SPECIFIC GRAVITY 1.003 - 1.035 >=1.030

METHOD: REFLECTANCE SPECTROPHOTOMETRY- PKA CHANGE OF AN IONIC POLYELECTROLYTE

BLOOD COUNTS, EDTA WHOLE BLOOD

HEMOGLOBIN	14.6	13.0 - 17.0	g/aL
METHOD: PHOTOMETRIC MEASUREMENT			
RED BLOOD CELL COUNT	5.79	High 4.5 - 5.5	mi l /μL

METHOD: COULTER PRINCIPLE WHITE BLOOD CELL COUNT 8.30 4.0 - 10.0 thou/µL

METHOD: COULTER PRINCIPLE PLATELET COUNT 320 150 - 410 thou/µL

METHOD: ELECTRONIC IMPEDENCE & MICROSCOPY

RBC AND PLATELET INDICES

HEMATOCRIT	45.9	40.0 - 50.0	%

METHOD: CALCULATED PARAMETER MEAN CORPUSCULAR VOL 79.3 Low 83.0 - 101.0 fL

METHOD: DERIVED PARAMETER FROM RBC HISTOGRAM

MEAN CORPUSCULAR HGB. Low 27.0 - 32.0 25.3 pg METHOD: CALCULATED PARAMETER

MEAN CORPUSCULAR HEMOGLOBIN 31.9 31 5 - 34 5 g/dL

CONCENTRATION METHOD: CALCULATED PARAMETER

MENTZER INDEX 13.7

RED CELL DISTRIBUTION WIDTH 14.7 **High** 11.6 - 14.0 % METHOD: DERIVED PARAMETER FROM RBC HISTOGRAM

MEAN PLATELET VOLUME 7.4 6.8 - 10.9

METHOD: DERIVED PARAMETER FROM PLATELET HISTOGRAM

CHEMICAL EXAMINATION, URINE

4.7 - 7.5

METHOD: REFLECTANCE SPECTROPHOTOMETRY- DOUBLE INDICATOR METHOD

NOT DETECTED NOT DETECTED

METHOD: REFLECTANCE SPECTROPHOTOMETRY - PROTEIN-ERROR-OF-INDICATOR PRINCIPLE

NOT DETECTED **GLUCOSE** NOT DETECTED

METHOD: REFLECTANCE SPECTROPHOTOMETRY, DOUBLE SEQUENTIAL ENZYME REACTION-GOD/POD





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KETONES		NOT DETECTED	NOT DETECTED	
METHOD: REFLECTANCE SP	PECTROPHOTOMETRY, ROTHERA'S PRINC	IPLE		
BLOOD		NOT DETECTED	NOT DETECTED	
METHOD : REFLECTANCE SF	PECTROPHOTOMETRY, PEROXIDASE LIKE	ACTIVITY OF HAEMOGLOBIN		
BILIRUBIN		NOT DETECTED	NOT DETECTED	
	PECTROPHOTOMETRY, DIAZOTIZATION- (
UROBILINOGEN		NORMAL	NORMAL	
	PECTROPHOTOMETRY - EHRLICH REACTION			
NITRITE		NOT DETECTED	NOT DETECTED	
	PECTROPHOTOMETRY, CONVERSION OF I			
LEUKOCYTE ESTERASE		NOT DETECTED	NOT DETECTED	
WBC DIFFERENTIAL	COUNT - NLR			
SEGMENTED NEUTROP	HILS	67	40 - 80	%
METHOD: VCSN TECHNOLO	OGY/ MICROSCOPY			
ABSOLUTE NEUTROPH	IL COUNT	5.56	2.0 - 7.0	thou/µL
METHOD : CALCULATED PAR	RAMETER			
LYMPHOCYTES		21	20 - 40	%
METHOD: VCSN TECHNOLO	OGY/ MICROSCOPY			
ABSOLUTE LYMPHOCYT	TE COUNT	1.74	1.0 - 3.0	thou/µL
METHOD : CALCULATED PAR				
NEUTROPHIL LYMPHOC	CYTE RATIO (NLR)	3.2		
METHOD : CALCULATED				
EOSINOPHILS		3	1.0 - 6.0	%
METHOD: VCSN TECHNOLO				
ABSOLUTE EOSINOPHI		0.25	0.02 - 0.50	thou/µL
METHOD : CALCULATED PAR	RAMETER			
MONOCYTES		9	2.0 - 10.0	%
METHOD: VCSN TECHNOLO		0.75	0.2.4.0	
ABSOLUTE MONOCYTE		0.75	0.2 - 1.0	thou/µL
METHOD : CALCULATED PAR	RAMETER	•	0 4	0.4
BASOPHILS		0	0 - 1	%
METHOD: VCSN TECHNOLO	·	0	0.03 0.10	kla a / l
ABSOLUTE BASOPHIL		0 Lo	w 0.02 - 0.10	thou/µL
METHOD : CALCULATED PAR				
MICROSCOPIC EXAM	IINATION, UKINE	2.2	0.5	(LIDE
PUS CELL (WBC'S)		2-3	0-5	/HPF
EPITHELIAL CELLS		0-1	0-5	/HPF
ERYTHROCYTES (RBC'S	S)	NOT DETECTED	NOT DETECTED	/HPF







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CASTS		NOT DETECTED		
CRYSTALS		NOT DETECTED		
BACTERIA		NOT DETECTED	NOT DETECTED	
YEAST		NOT DETECTED	NOT DETECTED	

METHOD: URINE ROUTINE & MICROSCOPY EXAMINATION BY INTEGRATED AUTOMATED SYSTEM

Comments

URINALYSIS: MICROSCOPIC EXAMINATION OF URINE IS CARRIED OUT ON CENTRIFUGED URINARY SEDIMENT.

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

ERYTHRO SEDIMENTATION RATE, BLOOD

SEDIMENTATION RATE (ESR)	4	0 - 14	mm at 1 hr
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METHOD: AUTOMATED (PHOTOMETRICAL CAPILLARY STOPPED FLOW KINETIC ANALYSIS)

GLUCOSE, FASTING, PLASMA

GLUCOSE, FASTING, PLASMA	98	74 - 99	mg/dL
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METHOD: SPECTROPHOTOMETRY HEXOKINASE

GLYCOSYLATED HEMOGLOBIN, EDTA WHOLE BLOOD

GLYCOSYLATED HEMOGLOBIN (HBA1C)	5.0	Non-diabetic: < 5.7	%

Pre-diabetics: 5.7 - 6.4 Diabetics: > or = 6.5ADA Target: 7.0 Action suggested: > 8.0

METHOD: ION-EXCHANGE HPLC

MEAN PLASMA GLUCOSE 96.8 < 116.0 mg/dL

METHOD: CALCULATED PARAMETER

GLUCOSE, POST-PRANDIAL, PLASMA

GLUCOSE, POST-PRANDIAL, PLASMA 97 70 - 139 mg/dL

METHOD: SPECTROPHOTOMETRY HEXOKINASE

CORONARY RISK PROFILE (LIPID PROFILE), SERUM

CHOLESTEROL High Desirable cholesterol level mg/dL 212

< 200

Borderline high cholesterol

200 - 239 High cholesterol > / = 240

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

TRIGLYCERIDES 66 Normal: < 150 mg/dL

Borderline high: 150 - 199 High: 200 - 499 Very High: >/= 500







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METHOD: SPECTROPHOTOMETRY, ENZYMATIC ENDPOIN	WITH GLYCFROL BLANK			
HDL CHOLESTEROL	60		Low HDL cholesterol	mg/dL
			< 40 High HDL cholesterol > / = 60	3, -
METHOD : SPECTROPHOTOMETRY, HOMOGENEOUS DIRE	CT ENZYMATIC COLORIMETRIC			
DIRECT LDL CHOLESTEROL	146	High	Optimal: < 100 Near optimal/above optimal: 129 Borderline high: 130 - 159 High: 160 - 189 Very high: > / = 190	mg/dL 100 -
METHOD : SPECTROPHOTOMETRY, HOMOGENEOUS ENZY	MATIC COLORIMETRIC			
NON HDL CHOLESTEROL METHOD: CALCULATED PARAMETER	152	High	Desirable : < 130 Above Desirable : 130 -159 Borderline High : 160 - 189 High : 190 - 219 Very high : > / = 220	mg/dL
CHOL/HDL RATIO	3,5		Low Risk : 3.3 - 4.4	
	5.5		Average Risk : 4.5 - 7.0 Moderate Risk : 7.1 - 11.0 High Risk : > 11.0	
METHOD : CALCULATED PARAMETER	2.4			
LDL/HDL RATIO	2.4		Desirable/Low Risk: 0.5 - 3.0 Borderline/Moderate Risk: 3.6.0 High Risk: > 6.0	
METHOD: CALCULATED PARAMETER				
VERY LOW DENSITY LIPOPROTEIN METHOD: CALCULATED PARAMETER	13.0		< or = 30.0	mg/dL
LIVER FUNCTION PROFILE, SERUM				
BILIRUBIN, TOTAL	0,62		Upto 1.2	mg/dL
METHOD : SPECTROPHOTOMETRY, COLORIMETRIC -DIAZ	O METHOD		·	<i>3.</i>
BILIRUBIN, DIRECT	0.29	High	0.0 - 0.2	mg/dL
METHOD: SPECTROPHOTOMETRY, JENDRASSIK & GROFF	- DIAZOTIZATION			2.
BILIRUBIN, INDIRECT	0.33		0.1 - 1.0	mg/dL
METHOD: CALCULATED PARAMETER				-
TOTAL PROTEIN	7.4		6.0 - 8.0	g/dL
METHOD: SPECTROPHOTOMETRY, COLORIMETRIC-BIUR	ET, REAGENT BLANK, SERUM BLAN	K		
ALBUMIN	4.8		3.97 - 4.94	g/dL
METHOD: SPECTROPHOTOMETRY, BROMOCRESOL GREEN	N(BCG) - DYE BINDING			
GLOBULIN	2.6		2.0 - 3.5	g/dL
METHOD: CALCULATED PARAMETER				



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AL DUMANI/CLODULIAN D	ATIO	1.0	10.21	DATIO
ALBUMIN/GLOBULIN RA		1.8	1.0 - 2.1	RATIO
METHOD : CALCULATED PAR		20	Units 40	1171
	NSFERASE (AST/SGOT)	20	Upto 40	U/L
	TETRY, WITHOUT PYRIDOXAL PHOSPH	35	Unto 41	U/L
ALANINE AMINOTRANS	FERASE (ALT/SGPT) 1ETRY, WITHOUT PYRIDOXAL PHOSPH		Upto 41	U/L
ALKALINE PHOSPHATAS		118	40 - 129	U/L
	IETRY, PNPP, AMP BUFFER - IFCC	110	40 - 129	U/L
GAMMA GLUTAMYL TRA	, ,	35	< 60	U/L
	, ,	G-GLUTAMYL-CARBOXY-NITROANILIDE		0/L
LACTATE DEHYDROGEN		204	< 232	U/L
	IETRY, LACTATE TO PYRUVATE - UV-IF		. 232	0/ L
SERUM BLOOD UREA				
BLOOD UREA NITROGE		7	6 - 20	mg/dL
	TETRY, UREASE -COLORIMETRIC	,	0 20	mg/aL
CREATININE, SERUM				
CREATININE		0.66 Lov	w 0,90 - 1,30	mg/dL
	1ETRY, JAFFE'S ALKALINE PICRATE KIN	IETIC - RATE BLANKED - IFCC-IDMS S		1119, 42
BUN/CREAT RATIO	,			
BUN/CREAT RATIO		10,60	8 - 15	
METHOD : CALCULATED PAR	AMETER	10100		
URIC ACID, SERUM				
URIC ACID		5.7	3.4 - 7.0	mg/dL
	METRY, ENZYMATIC COLORIMETRIC- U		311 710	mg/ aL
TOTAL PROTEIN, SER				
TOTAL PROTEIN		7.4	6.0 - 8.0	g/dL
	1ETRY, COLORIMETRIC -BIURET, REAG			g, aL
ALBUMIN, SERUM	,	,		
ALBUMIN		4.8	3,97 - 4,94	g/dL
	1ETRY, BROMOCRESOL GREEN(BCG) -		3137 1131	9, 42
GLOBULIN	,			
GLOBULIN		2,6	2.0 - 3.5	g/dL
METHOD : CALCULATED PAR	AMETER	210	210 313	9/ 42
ELECTROLYTES (NA/				
SODIUM	,,,	140	136 - 145	mmo l /L
METHOD : ISE INDIRECT		110	100 110	iiiiioi, L
POTASSIUM		4,10	3,5 - 5,1	mmo l /L
		.110	3.3 311	



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METHOD : ISE INDIRECT CHLORIDE 102 98 - 106 mmol/L METHOD : ISE INDIRECT THYROID PANEL, SERUM T3 162.0 80.0 - 200.0 ng/dL METHOD : COMPETITIVE ELECTROCHEMILUMINESCENCE IMMUNOASSAY T4 8.83 5.10 - 14.10 µg/dL METHOD : SANDWICH ELECTROCHEMILUMINESCENCE IMMUNOASSAY T5H 3RD GENERATION 1.280 0.270 - 4.200 µIU/mL METHOD : SANDWICH ELECTROCHEMILUMINESCENCE IMMUNOASSAY T5OOL: OVA & PARASITE COLOUR BROWN CONSISTENCY SEMI FORMED ODOUR FAECAL MUCUS NOT DETECTED NOT DETECTED MUCUS NOT DETECTED NOT DETECTED VISIBLE BLOOD ABSENT ABSENT POLYMORPHONUCLEAR LEUKOCYTES NOT DETECTED NOT DETECTED /HPF METHOD: MICROSCOPIC EXAMINATION RED BLOOD CELLS NOT DETECTED NOT DETECTED /HPF METHOD: MICROSCOPIC EXAMINATION RACROPHAGES NOT DETECTED NOT DETECTED /HPF METHOD: MICROSCOPIC EXAMINATION RACROPHAGES NOT DETECTED NOT DETECTED METHOD: MICROSCOPIC EXAMINATION ROTHOD: MICROSCOPIC EXAMINATION ROTHOD: MICROSCOPIC EXAMINATION ROTHOD: MICROSCOPIC EXAMINATION ROTHOD: MICROSCOPIC EXAMINATION LARVAE NOT DETECTED NOT DETECTED METHOD: MICROSCOPIC EXAMINATION LARVAE NOT DETECTED METHOD: MICROSCOPIC EXAMINATION ON DETECTED METHOD: MICROSCOPIC EXAMINATION MICROSCOPIC EXAMINATION NOT DETECTED METHOD: MICROSCOPIC EXAMINATION MICROSCOPIC EXAMINATION NOT DETECTED NOT DETECTED NOT DETECTED	Test Report Status <u>Final</u>		Results	Biological Reference Interval Units		
CHORIDE 102 98 - 106 mmol/L MEHIOD: ISE INDRECT THYROID PANEL, SERUM T3 162.0 80.0 - 200.0 ng/dL METHOD: COMPETITIVE ELECTROCHEMILUMINESCENCE IMMUNOASSIV TSM 38.83 5.10 - 14.10 µg/dL METHOD: COMPETITIVE ELECTROCHEMILUMINESCENCE IMMUNOASSIV TSM 38.83 5.10 - 14.10 µg/dL METHOD: COMPETITIVE ELECTROCHEMILUMINESCENCE IMMUNOASSIV TSM 38.83 5.10 - 14.10 µg/dL METHOD: COMPETITIVE ELECTROCHEMILUMINESCENCE IMMUNOASSIV TSM 38.83 5.10 - 14.10 µg/dL METHOD: SANDWICH ELECTROCHEMILUMINESCENCE IMMUNOASSIV TSM 38.83 5.10 - 14.10 µg/dL TSM 3D CENERATION 1.280 NOTA 4.200 µl/mL METHOD: SANDWICH ELECTROCHEMILUMINESCENCE IMMUNOASSIVE TSM 58.83 NOT DETECTED NOT DETECTED NOT DETECTED /HPF VISIBLE BLOOQ NOT DETECTED NOT DETECTED /HPF WETHOD: MEGROSCOPIC EXAMINATION NOT DETECTED NOT DETECTED						
### THYPROTO PANEL, SERUM T3 162.0 80.0 - 200.0 ng/dL ###################################			400	00 105	10	
THYROID PANEL, SERUM T3 162.0 80.0 - 200.0 ng/d. METHOD: COMPETITIVE ELECTROCHEMILUMINESCENCE IMMUNISSERVE			102	98 - 106	mmol/L	
T3 162.0 80.0 - 200.0 ng/dL METHOD: COMPETITIVE ELECTROCHEMILUMINESCENCE IMMUNOASSAY 8.83 5.10 - 14.10 μg/dL TSH 3RD GENERATION 1.280 0.270 - 4.200 μIU/mL METHOD: SANDWICH ELECTROCHEMILUMINESCENCE IMMUNOASSAY STOOL: OVA & PARASITE COLOUR BROWN COLOUR PAECAL COLOUR FAECAL MUCUS NOT DETECTED NOT DETECTED MUCUS NOT DETECTED NOT DETECTED MUCUS NOT DETECTED 0 - 5 /HPF METHOD: MICROSCOPIC EXAMINATION NOT DETECTED NOT DETECTED /HPF METHOD: MICROSCOPIC EXAMINATION NOT DETECTED						
METHOD : COMPETITIVE ELECTROCHEMILUMINESCENCE IMMUNOASSAY T4 8.83 5.10 - 14.10 µg/d. METHOD : COMPETITIVE ELECTROCHEMILUMINESCENCE IMMUNOASSAY TSH 3RD GENERATION 1.280 0.270 - 4.200 µIU/mL METHOD : SANDWICH ELECTROCHEMILUMINESCENCE IMMUNOASSAY STOOL: OVA & PARASITE COLOUR BROWN CONSISTENCY SEMI FORMED ODOUR FAECAL MUCUS NOT DETECTED NOT DETECTED WISIBLE BLOOD ABSENT ABSENT POLYMORPHONUCLEAR LEUKOCYTES NOT DETECTED 0 - 5 /HPF METHOD : MICROSCOPIC EXAMINATION MACROPHAGES NOT DETECTED NOT DETECTED /HPF METHOD : MICROSCOPIC EXAMINATION MACROPHAGES NOT DETECTED NOT DETECTED /HPF METHOD : MICROSCOPIC EXAMINATION CHARCOT-LEYDEN CRYSTALS NOT DETECTED NOT DETECTED METHOD : MICROSCOPIC EXAMINATION TROPHOZOITES NOT DETECTED NOT DETECTED METHOD : MICROSCOPIC EXAMINATION CYSTS NOT DETECTED NOT DETECTED METHOD : MICROSCOPIC EXAMINATION CYSTS NOT DETECTED NOT DETECTED METHOD : MICROSCOPIC EXAMINATION CYSTS NOT DETECTED NOT DETECTED METHOD : MICROSCOPIC EXAMINATION CYSTS NOT DETECTED METHOD : MICROSCOPIC EXAMINATION CYA METHOD : MICROSCOPIC EXAMINATION NOT DETECTED METHOD : MICROSCOPIC EXAMINATION LARVAE NOT DETECTED METHOD : MICROSCOPIC EXAMINATION MICROSCOPIC EXAMINATI	·	RUM				
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METHOD : COMPETITIVE ELECTROCHEMILUMINESCENCE IMMUNOASSAY TSH 3RD GENERATION 1.280 0.270 - 4.200 µIU/mL METHOD : SANDWICH ELECTROCHEMILUMINESCENCE IMMUNOASSAY STOOL: OVA & PARASITE COLOUR BROWN CONSISTENCY SEMI FORMED ODOUR FAECAL MUCUS NOT DETECTED NOT DETECTED VISIBLE BLOOD ABSENT ABSENT POLYMORPHONUCLEAR LEUKOCYTES NOT DETECTED NOT DETECTED METHOD: MICROSCOPIC EXMINATION RED BLOOD CELLS NOT DETECTED NOT DETECTED /HPF METHOD: MICROSCOPIC EXMINATION MACROPHAGES NOT DETECTED NOT DETECTED /HPF METHOD: MICROSCOPIC EXMINATION CHARCOT-LEYDEN CRYSTALS NOT DETECTED NOT DETECTED METHOD: MICROSCOPIC EXMINATION CYSTS NOT DETECTED NOT DETECTED METHOD: MICROSCOPIC EXMINATION CYSTS NOT DETECTED NOT DETECTED METHOD: MICROSCOPIC EXMINATION CYSTS NOT DETECTED METHOD: MICROSCOPIC EXMINATION CYA NOT DETECTED METHOD: MICROSCOPIC EXMINATION CYA NOT DETECTED METHOD: MICROSCOPIC EXMINATION LARVAE NOT DETECTED N		ECTROCHEMILUMINESCEN				
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ABO GROUP & RH TYPE, EDTA WHOLE BLOOD







ACROFEMI HEALTHCARE LTD (MEDIWHEEL)

F-703, LADO SARAI, MEHRAULI

SOUTH WEST DELHT **NEW DELHI 110030**

DELHI INDIA 8800465156

PLOT No. 88, ROAD No. 15, MIDC ESTATE, ANDHERI (EAST)

MUMBAI, 400093 MAHARASHTRA, INDIA

Tel: 09152729959/9111591115, Fax: CIN - U74899PB1995PLC045956

PATIENT NAME: SUJIT BHALERAO PATIENT ID: SUJIM535031940

ACCESSION NO: 0065VE001554 AGE: 33 Years SEX: Male

DRAWN: RECEIVED: 14/05/2022 07:42 REPORTED: 16/05/2022 14:11

REFERRING DOCTOR: SELF CLIENT PATIENT ID:

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

ABO GROUP 0

METHOD: HAEMAGGLUTINATION (AUTOMATED)

RH TYPE **POSITIVE**

METHOD: HAEMAGGLUTINATION (AUTOMATED)

XRAY-CHEST

IMPRESSION NO ABNORMALITY DETECTED

TMT OR ECHO

TMT OR ECHO ECHO DONE - NORMAL

ECG

ECG WITHIN NORMAL LIMITS

MEDICAL HISTORY

RELEVANT PRESENT HISTORY ITCHING AROUND NECK 2MONTHS

SCHIZOPHRENIA 2014.

RELEVANT PAST HISTORY **NOT SIGNIFICANT** RELEVANT PERSONAL HISTORY **NOT SIGNIFICANT**

RELEVANT FAMILY HISTORY ASTHMA.

HISTORY OF MEDICATIONS NOT SIGNIFICANT

ANTHROPOMETRIC DATA & BMI

HEIGHT IN METERS 1.59 mts 71 WEIGHT IN KGS. Kgs

BMI 28 BMI & Weight Status as follows: kg/sqmts

Below 18.5: Underweight 18.5 - 24.9: Normal 25.0 - 29.9: Overweight 30.0 and Above: Obese

GENERAL EXAMINATION

MENTAL / EMOTIONAL STATE **NORMAL** PHYSICAL ATTITUDE **NORMAL** GENERAL APPEARANCE / NUTRITIONAL STATUS **OBESE BUILT / SKELETAL FRAMEWORK AVERAGE** FACIAL APPEARANCE NORMAL SKIN **NORMAL** UPPER LIMB NORMAL LOWER LIMB **NORMAL NECK NORMAL**

NECK LYMPHATICS / SALIVARY GLANDS NOT ENLARGED OR TENDER

THYROID GLAND NOT ENLARGED







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8800465156

SRL Ltd

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

CAROTID PULSATION NORMAL BREAST (FOR FEMALES) NORMAL TEMPERATURE NORMAL

PULSE 70/MIN REGULAR, ALL PERIPHERAL PULSES WELL FELT

RESPIRATORY RATE NORMAL

CARDIOVASCULAR SYSTEM

BP 105/72 MM HG mm/Hg

(SUPINE) NORMAL NORMAL

HEART SOUNDS S1, S2 HEARD NORMALLY

MURMURS ABSENT

RESPIRATORY SYSTEM

SIZE AND SHAPE OF CHEST

MOVEMENTS OF CHEST

BREATH SOUNDS INTENSITY

NORMAL

BREATH SOUNDS QUALITY VESICULAR (NORMAL)

ADDED SOUNDS ABSENT

PER ABDOMEN

PERICARDIUM

APEX BEAT

APPEARANCE NORMAL VENOUS PROMINENCE ABSENT

LIVER NOT PALPABLE SPLEEN NOT PALPABLE HERNIA NORMAL

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

EYELIDS NORMAL **EYE MOVEMENTS** NORMAL CORNEA NORMAL DISTANT VISION RIGHT EYE WITHOUT GLASSES

WITHIN NORMAL LIMIT(6/6) DISTANT VISION LEFT EYE WITHOUT GLASSES WITHIN NORMAL LIMIT(6/6) NEAR VISION RIGHT EYE WITHOUT GLASSES WITHIN NORMAL LIMIT(N/6) NEAR VISION LEFT EYE WITHOUT GLASSES WITHIN NORMAL LIMIT(N/6) NORMAL

COLOUR VISION

BASIC ENT EXAMINATION

EXTERNAL EAR CANAL NORMAL TYMPANIC MEMBRANE **NORMAL**

NOSE NO ABNORMALITY DETECTED

SINUSES NORMAL

THROAT NO ABNORMALITY DETECTED

NOT ENLARGED TONSILS

SUMMARY

RELEVANT HISTORY ITCHING AROUND NECK 2MONTHS

SCHIZOPHRENIA 2014.

RELEVANT GP EXAMINATION FINDINGS OVERWEIGHT (HT: 159, WT: 71) RELEVANT LAB INVESTIGATIONS RAISED RED BLOOD CELL (5.79) RAISED DIRECT BILIRUBIN (0.29)

RAIED CHOLESTEROL (212)

RAISED DIRECT LDL CHOLESTEROL (146)

RELEVANT NON PATHOLOGY DIAGNOSTICS USG: MILD FATTY LIVER.

TINY BILATERAL RENAL CALCULI.

REMARKS / RECOMMENDATIONS LOW CALORIC DIET.

REDUCE FATTY FOOD IN DIET.

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 - NLRThe optimal threshold of 3.3 for NLR showed a prognostic possibility of clinical symptoms to change from mild to severe in COVID positive patients. When age = 49.5 years old and NLR = 3.3, 46.1% COVID-19 patients with mild disease might become severe. By contrast, when age < 49.5 years old and NLR < 3.3, COVID-19 patients tend to







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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 age, sex, menstrual cycle and drugs (eg. corticosteroids, contraceptives). It is especially low (0 -1mm) in polycythaemia, hypofibrinogenemia or congestive cardiac failure and when there are abnormalities of the red cells such as polikilocytosis, spherocytosis or sickle cells.

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

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:



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Tel: 09152729959/9111591115, Fax: CIN - U74899PB1995PLC045956

PATIENT NAME: SUJIT BHALERAO PATIENT ID: SUJIM535031940

0065VE001554 AGE: 33 Years SEX: Male ACCESSION NO:

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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, is chemia to the liver, chronic hepatitis, obstruction of bile ducts, cirrhosis.

ALP is a protein found in almost all body tissues. Tissues with higher amounts of ALP include the liver, bile ducts and bone. Elevated ALP levels are seen in Biliary obstruction,

Osteoblastic bone tumors, osteomalacia, hepatitis, Hyperparathyroidism, Leukemia, Lymphoma, Paget's disease, Rickets, Sarcoidosis etc. Lower-than-normal ALP levels seen in Hypophosphatasia, Malnutrition, Protein deficiency, Wilson's disease. GGT is an enzyme found in cell membranes of many tissues mainly in the liver, kidney and pancreas. It is also found in other tissues including intestine, spleen, heart, brain and seminal vesicles. The highest concentration is in the kidney, but the liver is considered the source of normal enzyme activity. Serum GGT has been widely used as an index of liver dysfunction. Elevated serum GGT activity can be found in diseases of the liver, 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 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:

- Mvasthenia 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



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• 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, SERUMSerum total protein, also known as total protein, is a biochemical test for measuring the total amount of protein in serum. Protein in the plasma is made up of albumin and

Higher-than-normal levels may be due to: Chronic inflammation or infection, including HIV and hepatitis B or C, Multiple myeloma, Waldenstrom's disease Lower-than-normal levels may be due to: Agammaglobulinemia, Bleeding (hemorrhage), Burns, Glomerulonephritis, Liver disease, Malabsorption, Malnutrition, Nephrotic syndrome, Protein-losing enteropathy etc.

ALBUMIN, SERUM-

Human serum albumin is the most abundant protein in human blood plasma. It is produced in the liver. Albumin constitutes about half of the blood serum protein. Low blood albumin levels (hypoalbuminemia) can be caused by: Liver disease like cirrhosis of the liver, nephrotic syndrome, protein-losing enteropathy, Burns, hemodilution, increased vascular permeability or decréased 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 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

respiratory actions, metabolic actions, metabolic actions, metabolic actions, feeling replications, metabolic actions, persistent gastic sectetion 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 (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) 0.1 - 2.5 0.2 - 3.0 0.3 - 3.0 81 - 190 100 - 260 First Trimester 2nd Trimester 6.6 - 12.4 6.6 - 15.5 3rd Trimester 6.6 - 15.5 100 - 260

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

(μg/dL) 1-3 day: 8.2 - 19.9 (ng/dL) New Born: 75 - 260 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.







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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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MEDI WHEEL FULL BODY HEALTH CHECK UP BELOW 40 MALE

ULTRASOUND ABDOMEN

ULTRASOUND ABDOMEN

MILD FATTY LIVER.TINY BILATERAL RENAL CALCULI.

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

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