



CLIENT CODE: C000138384 **CLIENT'S NAME AND ADDRESS:**

ACROFEMI HEALTHCARE LTD (MEDIWHEEL)

F-703, LADO SARAI, MEHRAULI SOUTH WEST DELHI **NEW DELHI 110030**

DELHI INDIA 8800465156

30-B, CHOWRINGEE MANSION, JAWAHARLAL NEHRU ROAD,

PATIENT ID:

KOLKATA, 700016 WEST BENGAL, INDIA

Tel: 033-22267333,46019048, Fax: 033-22271324

CIN - U74899PB1995PLC045956

PATIENT NAME: DEBABRATA BEHERA

DEBAF150474310

ACCESSION NO:

0082WB00080

AGE: 48 Years SEX: Male ABHA NO:

REPORTED:

01/03/2023 12:21

DRAWN: 25/02/2023 08:30

RECEIVED: 25/02/2023 08:47

CLIENT PATIENT ID:

Test Report Status

<u>Final</u>

Results

Biological Reference Interval Units

MEDI WHEEL FULL BODY HEALTH CHECK UP ABOVE 40 MALE

REFERRING DOCTOR: DR. ACROFEMI HEALTHCARE LTD (MEDIWHEEL)

RI OOD	COLINTS	FDTA	WHOL	E BLOOD

BLOOD COUNTS, LDTA WHOLL BLOOD			
HEMOGLOBIN (HB)	14.8	13.0 - 17.0	g/dL
METHOD: SPECTROPHOTOMETRY			
RED BLOOD CELL (RBC) COUNT	5.11	4.5 - 5.5	mil/μL
METHOD: ELECTRICAL IMPEDANCE			
WHITE BLOOD CELL (WBC) COUNT	5.19	4.0 - 10.0	thou/µL
METHOD: ELECTRICAL IMPEDANCE			
PLATELET COUNT	158	150 - 410	thou/µL
METHOD: ELECTRONIC IMPEDENCE & MICROSCOPY			
RBC AND PLATELET INDICES			
HEMATOCRIT (PCV)	43.3	40 - 50	%
METHOD : CALCULATED			
MEAN CORPUSCULAR VOLUME (MCV)	84.8	83 - 101	fL
METHOD: ELECTRICAL IMPEDANCE			
MEAN CORPUSCULAR HEMOGLOBIN (MCH)	29.0	27.0 - 32.0	pg
METHOD: CALCULATED			
MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION (MCHC) METHOD: CALCULATED	34.2	31.5 - 34.5	g/dL
RED CELL DISTRIBUTION WIDTH (RDW)	14.5	High 11.6 - 14.0	%
METHOD : ELECTRICAL IMPEDANCE			
MENTZER INDEX	16.6		
MEAN PLATELET VOLUME (MPV)	9.3	6.8 - 10.9	fL
METHOD : CALCULATED			
WBC DIFFERENTIAL COUNT			
NEUTROPHILS	59	40 - 80	%
METHOD: FLOWCYTOMETRY, ELECTRONIC IMPEDANCE & MIC	ROSCOPY.		
LYMPHOCYTES	31	20 - 40	%
METHOD: FLOWCYTOMETRY, ELECTRONIC IMPEDANCE & MIC	ROSCOPY.		
MONOCYTES	8	2 - 10	%
METHOD: FLOWCYTOMETRY, ELECTRONIC IMPEDANCE & MIC	ROSCOPY.		
EOSINOPHILS	2	1 - 6	%
BASOPHILS	0	0 - 2	%
METHOD: FLOWCYTOMETRY, ELECTRONIC IMPEDANCE & MIC	ROSCOPY.		
ABSOLUTE NEUTROPHIL COUNT	3.06	2.0 - 7.0	thou/µL
METHOD: FLOWCYTOMETRY & CALCULATED			



Page 1 Of 18 Scan to View Report





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KOLKATA, 700016 WEST BENGAL, INDIA

Tel: 033-22267333,46019048, Fax: 033-22271324 CIN - U74899PB1995PLC045956

 $\textbf{High} \ < 116.0$

PATIENT NAME: DEBABRATA BEHERA PATIENT ID: **DEBAF150474310**

0082WB00080 AGE: 48 Years ACCESSION NO: SEX: Male ABHA NO:

DRAWN: 25/02/2023 08:30 RECEIVED: 25/02/2023 08:47 01/03/2023 12:21 REPORTED:

REFERRING DOCTOR: DR. ACROFEMI HEALTHCARE LTD (MEDIWHEEL) CLIENT PATIENT ID:

Test Report Status <u>Final</u>	Results		Biological Reference Interva	l Units
ABSOLUTE LYMPHOCYTE COUNT	1.61		1 - 3	thou/µL
METHOD: FLOWCYTOMETRY & CALCULATED ABSOLUTE MONOCYTE COUNT METHOD: FLOWCYTOMETRY & CALCULATED	0.42		0.20 - 1.00	thou/µL
ABSOLUTE EOSINOPHIL COUNT METHOD : FLOWCYTOMETRY & CALCULATED	0.10		0.02 - 0.50	thou/µL
ABSOLUTE BASOPHIL COUNT METHOD: FLOWCYTOMETRY & CALCULATED	0	Low	0.02 - 0.10	thou/µL
MORPHOLOGY				
RBC METHOD: MICROSCOPIC EXAMINATION	NORMOCYTIC NO	ORMOCHRO	MIC	
WBC	NORMAL MORPH	HOLOGY		
METHOD: MICROSCOPIC EXAMINATION				
PLATELETS METHOD: MICROSCOPIC EXAMINATION	ADEQUATE			
ERYTHROCYTE SEDIMENTATION RATE (ESR),WHOLE			
BLOOD E.S.R	6		0 - 14	mm at 1 hr
METHOD: AUTOMATED (PHOTOMETRICAL CAPILLARY STO			0 14	min at 1 m
GLYCOSYLATED HEMOGLOBIN(HBA1C), BLOOD	,			
HBA1C	6.9	High	Non-diabetic Adult < 5.7 Pre-diabetes 5.7 - 6.4 Diabetes diagnosis: > or = 6.5 Therapeutic goals: < 7.0 Action suggested: > 8.0 (ADA Guideline 2021)	%
METHOD: HPLC			,	

151.3



ESTIMATED AVERAGE GLUCOSE(EAG)



mg/dL

Page 2 Of 18





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> SRL LIMITED - KOLKATA REF. LAB Bio-Rad Variant II Turbo CDM 5.4 S/N: 16043

> > 8213864667

PATIENT REP V2TURBO_A1c

Patient Data

Sample ID: Patient ID:

Name: Physician: Sex

DOB:

Comments:

A	h i-	Data
Апа	IVSIS	Data

Analysis Performed: 25/FEB/2023 15:02:07

Injection Number: 6866 Run Number: 317 Rack ID: 0004 Tube Number: 6

Report Generated: 25/FEB/2023 15:57:20

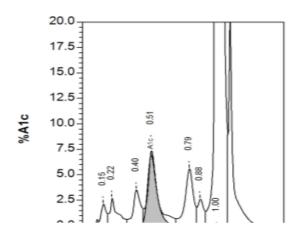
Operator ID:

Peak Name	NGSP %	Area %	Retention Time (min)	Peak Area
A1a		1.0	0.154	16982
A1b		1.8	0.218	31811
LA1c		2.1	0.403	36126
A1c	6.9*		0.512	98314
P3		4.0	0.794	68750
P4		1.3	0.875	23072
Ao		84.1	1.001	1450721

^{*}Values outside of expected ranges

Total Area: 1,725,776

HbA1c (NGSP) = 6.9* %





Page 3 Of 18 Scan to View Report





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Results

Biological Reference Interval Units

Comments

FOR HhA1C

NOTE: INCREASED LEVELS OF GLYCOSYLATED HEMOGLOBIN MAY NEED CLINICAL CORRELATION . HIGH GLYCOSYLATED HEMOGLOBIN LEVELS MAY BE OBSERVED IN CONDITIONS SUCH AS UNCONTROLLED DIABETES, POOR COMPLIANCE WITH ANTIDIABETIC THERAPY, CHRONIC RENAL FAILURE, HYPERTRIGLYCERIDEMIA, IRON DEFICIENCY ANAEMIA, SALICYLATE THERAPY, HAEMOGLOBINOPATHIES LIKE THALASSAEMIA MAY ALSO SHOW HIGH GLYCOSYLATED HEMOGLOBIN LEVELS.

GLUCOSE FASTING, FLUORIDE PLASMA

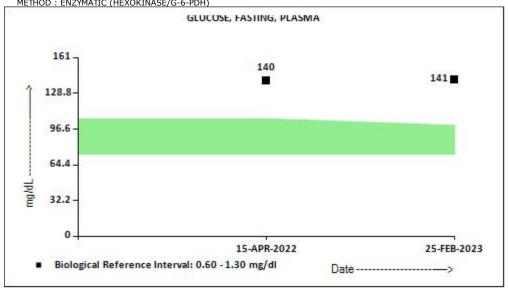
FBS (FASTING BLOOD SUGAR)

141

High 74 - 100

mg/dL

METHOD: ENZYMATIC (HEXOKINASE/G-6-PDH)



GLUCOSE, POST-PRANDIAL, PLASMA

PPBS(POST PRANDIAL BLOOD SUGAR) 191 High 140 Normal mg/dL 140 - 199 Pre-diabetic

> or = 200 Diabetic

METHOD: ENZYMATIC (HEXOKINASE/G-6-PDH)

LIPID PROFILE, SERUM

CHOLESTEROL, TOTAL 187

< 200 Desirable

mg/dL

200 - 239 Borderline High

>/= 240 High

mg/dL

METHOD: ENZYMATIC ASSAY

TRIGLYCERIDES 133

< 150 Normal 150 - 199

Borderline High 200 - 499 High

>/=500 Very High



Page 4 Of 18





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ACCESSION NO:

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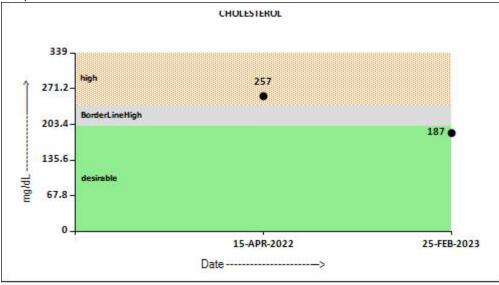
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Test Report Status <u>Final</u>	Results		Biological Reference Inter	val Units
METHOD : GLYCEROL PHOSPHATE OXIDASE HDL CHOLESTEROL	37	Low	Low: < 40	mg/dL
METHOD: ACCELERATOR SELECTIVE DETERGENT METHODOLOGY			High: $ > / = 60 $	
CHOLESTEROL LDL	123			mg/dL
NON HDL CHOLESTEROL	150	High	Desirable: Less than 130 Above Desirable: 130-159 Borderline High: 160-189 High: 190 -219 Very High: >or = 220	mg/dL
METHOD: CALCULATED				
VERY LOW DENSITY LIPOPROTEIN	26.6			mg/dL
CHOL/HDL RATIO	5.1			
LDL/HDL RATIO	3.3			











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

Interpretation(s)

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

Serum lipid profile is measured for cardiovascular risk prediction. 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			
		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 Hypercholesterolemi	a		
High Risk	1. Three major ASCVD risk factors. 2. Dia	abetes with 1 major risk factor or no evidence of end		
		organ damage. 3. CKD stage 3B or 4. 4. LDL >190 mg/dl 5. Extreme of a single risk factor. 6.		
	Coronary Artery Calcium - CAC >300 AU.	Coronary Artery Calcium - CAC >300 AU. 7. Lipoprotein a >/= 50mg/dl 8. Non stenotic carotid		
	plaque			
Moderate Risk	2 major ASCVD risk factors	2 major ASCVD risk factors		
Low Risk	0-1 major ASCVD risk factors	0-1 major ASCVD risk factors		
Major ASCVD (Ath	therosclerotic cardiovascular disease) Risk Factors			
1. Age $>$ or $=$ 45 year	s in males and > or = 55 years in females 3. Current Cigarette smoking or tobacco use			
2. Family history of p	emature ASCVD 4. High blood pressure			
5. Low HDL				

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

Risk Group	Treatment Goals		Consider Drug Thera	py
	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)		









Units

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Biological Reference Interval

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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				
Vory High Rick	<50	<80	>OP- 50	>OP - 80

Results

Extreme Risk Group	$\langle OR = 30$	<OR = 60	> 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.

Final

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	1.65	High	0.2 - 1.2	mg/dL
METHOD : DIAZONIUM SALT		_		
BILIRUBIN, DIRECT	0.51	High	0.0 - 0.5	mg/dL
METHOD : DIAZO REACTION				<i>3.</i>
BILIRUBIN, INDIRECT	1.14	High	0.1 - 1.0	mg/dL
METHOD : CALCULATED				
TOTAL PROTEIN	7.1		6.0 - 8.30	g/dL
METHOD : BIURET				
ALBUMIN	4.8		3.5 - 5.2	g/dL
METHOD: COLORIMETRIC (BROMCRESOL GREEN)				
GLOBULIN	2.3		2.0 - 3.5	g/dL
ALBUMIN/GLOBULIN RATIO	2.1		1 - 2.1	RATIO
METHOD : CALCULATED PARAMETER				
ASPARTATE AMINOTRANSFERASE (AST/SGOT)	34		5 - 34	U/L
METHOD: ENZYMATIC (NADH (WITHOUT P-5'-P)				
ALANINE AMINOTRANSFERASE (ALT/SGPT)	55		0 - 55	U/L
METHOD: ENZYMATIC (NADH (WITHOUT P-5'-P)				
ALKALINE PHOSPHATASE	60		40 - 150	U/L
METHOD: PARA-NITROPHENYL PHOSPHATE				
GAMMA GLUTAMYL TRANSFERASE (GGT)	21		11 - 59	U/L
METHOD: L-GAMMA-GLUTAMYL-4-NITROANALIDE/GLYCYLGLYCINE K	CINETIC METHOD			
LACTATE DEHYDROGENASE	139		125 - 220	U/L
METHOD : IFCC LACTATE TO PYRUVATE				
BLOOD UREA NITROGEN (BUN), SERUM				
BLOOD UREA NITROGEN	9		8.9 - 20.6	mg/dL
METHOD : UREASE METHOD				
CREATININE, SERUM				
CREATININE	0.89		0.60 - 1.30	mg/dL



Page 7 Of 18 Scan to View Report





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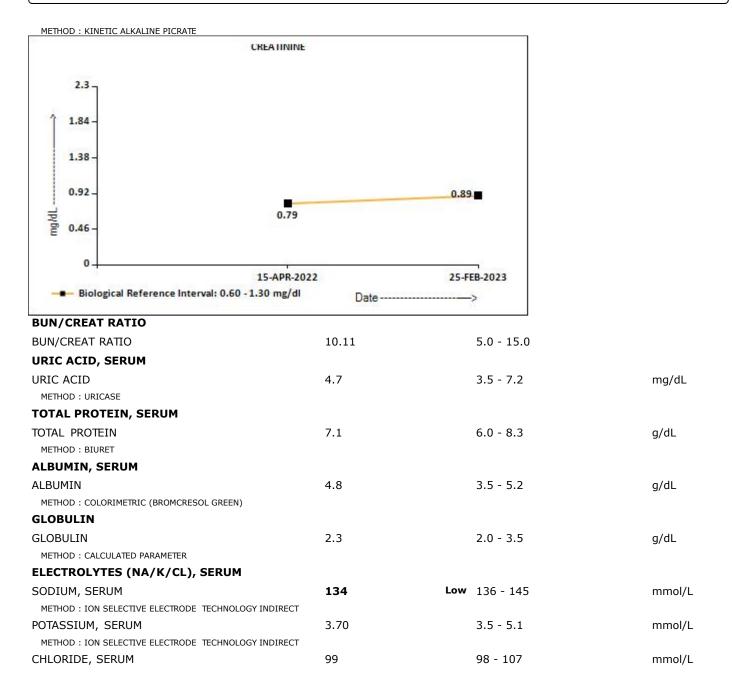
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METHOD: ION SELECTIVE ELECTRODE TECHNOLOGY INDIRECT

Interpretation(s)

Sodium	Potassium	Chloride
Decreased in:CCF, cirrhosis,	Decreased in: Low potassium	Decreased in: Vomiting, diarrhea,
vomiting, diarrhea, excessive	intake, prolonged vomiting or diarrhea,	renal failure combined with salt
sweating, salt-losing	RTA types I and II,	deprivation, over-treatment with
nephropathy, adrenal insufficiency,	hyperaldosteronism, Cushing's	diuretics, chronic respiratory acidosis
nephrotic syndrome, water	syndrome,osmotic diuresis (e.g.,	diabetic ketoacidosis, excessive
intoxication, SIADH. Drugs:	hyperglycemia),alkalosis, familial	sweating, SIADH, salt-losing
thiazides, diuretics, ACE inhibitors,	periodic paralysis,trauma	nephropathy, porphyria, expansion of
chlorpropamide, carbamazepine, anti	(transient).Drugs: Adrenergic agents,	extracellular fluid volume,
depressants (SSRI), antipsychotics.	diuretics.	adrenalinsufficiency,
, , , , , , , , , , , , , , , , , , , ,		hyperaldosteronism, metabolic
		alkalosis. Drugs: chronic
		laxative,corticosteroids, diuretics.
Increased in: Dehydration	Increased in: Massive hemolysis,	Increased in: Renal failure, nephrotic
(excessivesweating, severe	severe tissue damage, rhabdomyolysis,	syndrome, RTA, dehydration,
vomiting or diarrhea), diabetes	acidosis, dehydration,renal failure,	overtreatment with
mellitus, diabetesinsipidus,	Addison's disease, RTA type IV,	saline,hyperparathyroidism, diabetes
hyperaldosteronism, inadequate	hyperkalemic familial periodic	insipidus, metabolic acidosis from
water intake. Drugs: steroids,	paralysis. Drugs: potassium salts,	diarrhea (Loss of HCO3-), respiratory
licorice, oral contraceptives.	potassium- sparing diuretics,NSAIDs,	alkalosis, hyperadre no corticism.
	beta-blockers, ACE inhibitors, high-	Drugs: acetazolamide, androgens,
	dose trimethoprim-sulfamethoxazole.	hydrochlorothiazide, salicylates.
Interferences: Severe lipemia or	Interferences: Hemolysis of sample,	Interferences:Test is helpful in
hyperproteinemi, if sodium analysis	delayed separation of serum,	assessing normal and increased anion
involves a dilution step can cause	prolonged fist clenching during blood	gap metabolic acidosis and in
spurious results. The serum sodium	drawing, and prolonged tourniquet	distinguishing hypercalcemia due to
falls about 1.6 mEq/L for each 100	placement. Very high WBC/PLT counts	hyperparathyroidism (high serum
mg/dL increase in blood glucose.	may cause spurious. Plasma potassium	chloride) from that due to malignancy
	levels are normal.	(Normal serum chloride)

PHYSICAL EXAMINATION, URINE

COLOR	PALE YELLOW	
APPEARANCE	CLEAR	
CHEMICAL EXAMINATION, URINE		
PH	6.0	4.7 - 7.5
SPECIFIC GRAVITY	1.005	1.003 - 1.035
METHOD: DIPSTICK		
PROTEIN	NOT DETECTED	NOT DETECTED
METHOD: DIPSTICK		
GLUCOSE	NOT DETECTED	NOT DETECTED
METHOD : DIPSTICK		
KETONES	NOT DETECTED	NOT DETECTED
METHOD : DIPSTICK		
BLOOD	NOT DETECTED	NOT DETECTED
METHOD : DIPSTICK		
BILIRUBIN	NOT DETECTED	NOT DETECTED









CLIENT CODE: C000138384 **CLIENT'S NAME AND ADDRESS:**

ACROFEMI HEALTHCARE LTD (MEDIWHEEL)

F-703, LADO SARAI, MEHRAULI

SOUTH WEST DELHI **NEW DELHI 110030 DELHI INDIA**

30-B, CHOWRINGEE MANSION, JAWAHARLAL NEHRU ROAD, KOLKATA, 700016

PATIENT ID:

WEST BENGAL, INDIA

Tel: 033-22267333,46019048, Fax: 033-22271324

CIN - U74899PB1995PLC045956

PATIENT NAME: DEBABRATA BEHERA

DEBAF150474310

ACCESSION NO:

8800465156

0082WB00080

<u>Final</u>

AGE: 48 Years SEX: Male ABHA NO: REPORTED:

01/03/2023 12:21

DRAWN: 25/02/2023 08:30

RECEIVED: 25/02/2023 08:47

REFERRING DOCTOR: DR. ACROFEMI HEALTHCARE LTD (MEDIWHEEL) CLIENT PATIENT ID:

> **Biological Reference Interval** Units

METHOD : DIPSTICK

Test Report Status

UROBILINOGEN METHOD : DIPSTICK

NORMAL

Results

NORMAL

NOT DETECTED

NOT DETECTED

METHOD : DIPSTICK

LEUKOCYTE ESTERASE

NEGATIVE

NOT DETECTED

MICROSCOPIC EXAMINATION, URINE

RED BLOOD CELLS PUS CELL (WBC'S)

NOT DETECTED 1-2

NOT DETECTED 0-5

/HPF

EPITHELIAL CELLS

1-2

0-5

/HPF /HPF

CASTS

NITRITE

NOT DETECTED

NOT DETECTED

CRYSTALS BACTERIA

YEAST

NOT DETECTED NOT DETECTED NOT DETECTED

NOT DETECTED

Comments

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









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WEST BENGAL, INDIA

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

Interpretation(s)

The following table describes the probable conditions, in which the analytes are present in urine

Presence of	Conditions		
Proteins	Inflammation or immune illnesses		
Pus (White Blood Cells)	Urinary tract infection, urinary tract or kidney stone, tumors or any kind		
	of kidney impairment		
Glucose	Diabetes or kidney disease		
Ketones	Diabetic ketoacidosis (DKA), starvation or thirst		
Urobilinogen	Liver disease such as hepatitis or cirrhosis		
Blood	Renal or genital disorders/trauma		
Bilirubin	Liver disease		
Erythrocytes	Urological diseases (e.g. kidney and bladder cancer, urolithiasis), urinary		
	tract infection and glomerular diseases		
Leukocytes	Urinary tract infection, glomerulonephritis, interstitial nephritis either		
	acute or chronic, polycystic kidney disease, urolithiasis, contamination by		
	genital secretions		
Epithelial cells	Urolithiasis, bladder carcinoma or hydronephrosis, ureteric stents or		
	bladder catheters for prolonged periods of time		
Granular Casts	Low intratubular pH, high urine osmolality and sodium concentration,		
	interaction with Bence-Jones protein		
Hyaline casts	Physical stress, fever, dehydration, acute congestive heart failure, renal		
	diseases		
Calcium oxalate	Metabolic stone disease, primary or secondary hyperoxaluria, intravenous		
	infusion of large doses of vitamin C, the use of vasodilator naftidrofuryl		
	oxalate or the gastrointestinal lipase inhibitor orlistat, ingestion of		
	ethylene glycol or of star fruit (Averrhoa carambola) or its juice		
Uric acid	arthritis		
Bacteria	Urinary infectionwhen present in significant numbers & with pus cells.		
Trichomonas vaginalis	Vaginitis, cervicitis or salpingitis		

THYROID PANEL, SERUM

T3	112.5	35 - 193	ng/dL
METHOD: TWO-STEP CHEMILUMINESCENT MICROPARTIC	CLE IMMUNOASSAY		
T4	8.67	4.87 - 11.71	μg/dL
METHOD: TWO-STEP CHEMILUMINESCENT MICROPARTIC	CLE IMMUNOASSAY		
TSH (ULTRASENSITIVE)	1.149	0.350 - 4.940	μIU/mL

METHOD: TWO-STEP CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY









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

Interpretation(s)

Triiodothyronine T3, **Thyroxine T4**, and **Thyroid Stimulating Hormone TSH** are thyroid hormones which affect 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.

Excessive secretion of thyroxine in the body is hyperthyroidism, and deficient secretion is called hypothyroidism.

In primary hypothyroidism, TSH levels are significantly elevated, while in secondary and tertiary hyperthyroidism, TSH levels are low. Below mentioned are the guidelines for Pregnancy related reference ranges for Total T4, TSH & Total T3. Measurement of the serum TT3 level is a more sensitive test for the diagnosis of hyperthyroidism, and measurement of TT4 is more useful in the diagnosis of 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. It is advisable to detect Free T3, FreeT4 along with TSH, instead of testing for albumin bound Total T3, Total T4.

Sr. No.	TSH	Total T4	FT4	Total T3	Possible Conditions
1	High	Low	Low	Low	(1) Primary Hypothyroidism (2) Chronic autoimmune Thyroiditis (3)
					Post Thyroidectomy (4) Post Radio-Iodine treatment
2	High	Normal	Normal	Normal	(1)Subclinical Hypothyroidism (2) Patient with insufficient thyroid hormone replacement therapy (3) In cases of Autoimmune/Hashimoto thyroiditis (4). Isolated increase in TSH levels can be due to Subclinical inflammation, drugs like amphetamines, Iodine containing drug and dopamine antagonist e.g. domperidone and other physiological reasons.
3	Normal/Low	Low	Low	Low	(1) Secondary and Tertiary Hypothyroidism
4	Low	High	High	High	(1) Primary Hyperthyroidism (Graves Disease) (2) Multinodular Goitre (3)Toxic Nodular Goitre (4) Thyroiditis (5) Over treatment of thyroid hormone (6) Drug effect e.g. Glucocorticoids, dopamine, T4 replacement therapy (7) First trimester of Pregnancy
5	Low	Normal	Normal	Normal	(1) Subclinical Hyperthyroidism
6	High	High	High	High	(1) TSH secreting pituitary adenoma (2) TRH secreting tumor
7	Low	Low	Low	Low	(1) Central Hypothyroidism (2) Euthyroid sick syndrome (3) Recent treatment for Hyperthyroidism
8	Normal/Low	Normal	Normal	High	(1) T3 thyrotoxicosis (2) Non-Thyroidal illness
9	Low	High	High	Normal	(1) T4 Ingestion (2) Thyroiditis (3) Interfering Anti TPO antibodies

REF: 1. TIETZ Fundamentals of Clinical chemistry 2.Guidlines of the American Thyroid association during pregnancy and Postpartum, 2011. **NOTE: It is advisable to detect Free T3,FreeT4 along with TSH, instead of testing for albumin bound Total T3, Total T4.**TSH is not affected by variation in thyroid - binding protein. TSH has a diurnal rhythm, with peaks at 2:00 - 4:00 a.m. And troughs at 5:00 - 6:00 p.m. With ultradian variations.

PHYSICAL EXAMINATION, STOOL

COLOUR SAMPLE NOT RECEIVED

METHOD : VISUAL

ABO GROUP & RH TYPE, EDTA WHOLE BLOOD

ABO GROUP TYPE O

METHOD : GEL CARD METHOD

RH TYPE POSITIVE









DEBAF150474310

Units

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CIN - U74899PB1995PLC045956

PATIENT NAME: DEBABRATA BEHERA

0082WB00080

<u>Final</u>

AGE: 48 Years SEX: Male ABHA NO:

Test Report Status

ACCESSION NO:

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Results

REPORTED: 01/03/2023 12:21

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CLIENT PATIENT ID:

Biological Reference Interval

METHOD: GEL CARD METHOD

XRAY-CHEST

IMPRESSION

NO SIGNIFICANT ABNORMALITY DETECTED

TMT OR ECHO

TMT OR ECHO

ECHO DONE INSTEAD OF TMT

IMPRESSION-Normal Study.

ECG

FCG

WITHIN NORMAL LIMITS

MEDICAL HISTORY

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

RELEVANT FAMILY HISTORY FATHER: BRONCHIAL ASTHMA

OCCUPATIONAL HISTORY NOT SIGNIFICANT HISTORY OF MEDICATIONS NOT SIGNIFICANT

ANTHROPOMETRIC DATA & BMI

HEIGHT IN METERS 1.63 mts WEIGHT IN KGS. 64 Kgs

BMI 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

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

NECK LYMPHATICS / SALIVARY GLANDS NOT ENLARGED OR TENDER

THYROID GLAND **NOT ENLARGED**

CAROTID PULSATION **NORMAL**









DEBAF150474310

mm/Hg

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Test Report Status Biological Reference Interval Results Units <u>Final</u> **NORMAL TEMPERATURE PULSE** 80/MINS RESPIRATORY RATE NORMAL **CARDIOVASCULAR SYSTEM**

ΒP 134/70

PERICARDIUM NORMAL

NORMAL APEX BEAT

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**

CENTRAL NERVOUS SYSTEM

HIGHER FUNCTIONS **NORMAL** CRANIAL NERVES NORMAL CEREBELLAR FUNCTIONS **NORMAL** SENSORY SYSTEM **NORMAL** MOTOR SYSTEM **NORMAL REFLEXES NORMAL**

MUSCULOSKELETAL SYSTEM

SPINE NORMAL JOINTS NORMAL

BASIC EYE EXAMINATION

CONJUNCTIVA **NORMAL EYELIDS** NORMAL EYE MOVEMENTS NORMAL



Page 14 Of 18 Scan to View Report





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CIN - U74899PB1995PLC045956

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0082WB00080

48 Years AGE:

SEX: Male

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Test Report Status

<u>Final</u>

Results

Biological Reference Interval Units

DISTANT VISION RIGHT EYE WITHOUT GLASSES 6/18 DISTANT VISION LEFT EYE WITHOUT GLASSES 6/15 NEAR VISION RIGHT EYE WITHOUT GLASSES N9 NEAR VISION LEFT EYE WITHOUT GLASSES N9 COLOUR VISION NORMAL

BASIC ENT EXAMINATION

EXTERNAL EAR CANAL **NORMAL** TYMPANIC MEMBRANE NORMAL

NOSE NO ABNORMALITY DETECTED

SINUSES NORMAI

THROAT NO ABNORMALITY DETECTED

TONSILS NOT ENLARGED

BASIC DENTAL EXAMINATION

TEETH NORMAL GUMS HEALTHY

SUMMARY

REMARKS / RECOMMENDATIONS

Mr. BEHERA CAME FOR ANNUAL HEALTH CHECK UP. ON EXAMINATION AND INVESTIGATIONS HE IS FOUND TO HAVE RAISED FBS(141mg%)

<PPBS(191mg%) & BILIRUBIN(1.65mg%).

1. DIET AS DISCUSSED.

2. REGULAR PHYSICAL EXERCISE AND WALKING.

3. DRINK PLENTY OF WATER.

4. VISION CORRECTION

5. CONSULT COMPANY MEDICAL OFFICER/FAMILY PHYSICIAN

Comments

MEDICAL EXAMINATION DONE BY: DR. B. N. JANA, MBBS, DCH CONSULTANT WELLNESS CLINIC PARK STREET, KOLKATA

Interpretation(s)

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

RBC AND PLATELET INDICES-Mentzer index (MCV/RBC) is an automated cell-counter based calculated screen tool to differentiate cases of Iron deficiency anaemia(>13) from Beta thalassaemia trait

(<13) in patients with microcytic anaemia. This needs to be interpreted in line with clinical correlation and suspicion. Estimation of HbA2 remains the gold standard for diagnosing a case of beta thalassaemia trait.





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SRL Ltd

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WBC DIFFERENTIAL COUNT-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.

ERYTHROCYTE SEDIMENTATION RATE (ESR), WHOLE BLOOD-**TEST DESCRIPTION**:Erythrocyte sedimentation rate (ESR) is a test that indirectly measures the degree of inflammation present in the body. The test actually measures the rate of fall (sedimentation) of erythrocytes in a sample of blood that has been placed into a tall, thin, vertical tube. Results are reported as the millimetres of clear fluid (plasma) that are present at the top portion of the tube after one hour. Nowadays fully automated instruments are available to measure ESR.

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

TEST INTERPRETATION

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

Estrogen medication, Aging.
Finding a very accelerated ESR(>100 mm/hour) in patients with ill-defined symptoms directs the physician to search for a systemic disease (Paraproteinemias,

Disseminated malignancies, connective tissue disease, severe infections such as bacterial endocarditis).

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

Decreased in: Polycythermia vera, Sickle cell anemia

LIMITATIONS

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

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

salicylates)

1. Nathan and Oski's Haematology of Infancy and Childhood, 5th edition 2. Paediatric reference intervals. AACC Press, 7th edition. Edited by S. Soldin 3. The reference for the adult reference range is "Practical Haematology by Dacie and Lewis,10th edition.

GLYCOSYLATED HEMOGLOBIN(HBA1C), EDTA WHOLE BLOOD-**Used For**:

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

The ADA recommends measurement of HbA1c (typically 3-4 times per year for type 1 and poorly controlled type 2 diabetic patients, and 2 times per year for well-controlled type 2 diabetic patients) to determine whether a patients metabolic control has remained continuously within the target range. 1.eAG (Estimated average glucose) converts percentage HbA1c to md/dl, to compare blood glucose levels.

- 2. eAG gives an evaluation of blood glucose levels for the last couple of months.
 3. eAG is calculated as eAG (mg/dl) = 28.7 * HbA1c 46.7

HbA1c Estimation can get affected due to :I.Shortened Erythrocyte survival : Any condition that shortens erythrocyte survival or decreases mean erythrocyte age (e.g. recovery from acute blood loss, hemolytic anemia) will falsely lower HbA1c test results. Fructosamine is recommended in these patients which indicates diabetes control over 15 days. II. Vitamin C & E are reported to falsely lower test results. (possibly by inhibiting glycation of hemoglobin.

III.Iron deficiency anemia is reported to increase test results. Hypertriglyceridemia, uremia, hyperbilirubinemia, chronic alcoholism,chronic ingestion of salicylates & opiates addiction are reported to interfere with some assay methods, falsely increasing results. IV.Interference of hemoglobinopathies in HbA1c estimation is seen in

a.Homozygous hemoglobinopathy. Fructosamine is recommended for testing of HbA1c. b.Heterozygous state detected (D10 is corrected for HbS & HbC trait.)

c.HbF > 25% on alternate paltform (Boronate affinity chromatography) is recommended for testing of HbA1c.Abnormal Hemoglobin electrophoresis (HPLC method) is recommended for detecting a hemoglobinopathy GLUCOSE FASTING,FLUORIDE PLASMA-**TEST DESCRIPTION**

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

Diabetes mellitus, Cushing's syndrome (10 - 15%), chronic pancreatitis (30%). Drugs:corticosteroids,phenytoin, estrogen, thiazides.

Pancreatic islet cell disease with increased insulin,insulinoma,adrenocortical insufficiency, hypopituitarism,diffuse liver disease, malignancy (adrenocortical, ranticatic isac cen usease with incleased insulin,insulinona, an encountral insulinierity, hypopituicalishi, dinuse in stomach, fibrosarcoma), infant of a diabetic mother, enzyme deficiency diseases(e.g., galactosemia), Drugs- insulin, ethanol, propranolol sulfonylureas, tolbutamide, and other oral hypoglycemic agents.

NOTE: While random serum glucose levels correlate with home glucose monitoring results (weekly mean capillary glucose values), there is wide fluctuation within individuals. Thus, glycosylated hemoglobin(HbA1c) levels are favored to monitor glycemic control.

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









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

GLUCOSE, POST-PRANDIAL, PLASMA-High fasting glucose level in comparison to post prandial glucose level may be seen due to effect of Oral Hypoglycaemics & Insulin treatment, Renal Glyosuria, Glycaemic index & response to food consumed, Alimentary Hypoglycemia, Increased insulin response & sensitivity etc.Additional test HbA1c LIVER FUNCTION PROFILE, SERUM-LIVER FUNCTION PROFILE

Bilirubin is a yellowish pigment found in bile and is a breakdown product of normal heme catabolism. Bilirubin is excreted in bile and urine, and elevated levels may give yellow discoloration in jaundice. Elevated levels results from increased bilirubin production (eg, hemolysis and ineffective erythropoiesis), decreased bilirubin excretion (eg, obstruction and hepatitis), and abnormal bilirubin metabolism (eg, herinding) fundice). 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.

nepatitis, obstruction of bile ducts, cirrnosis.

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

BLOOD UREA NITROGEN (BUN), SERUM-Causes of Increased levels include Pre renal (High protein diet, Increased protein catabolism, GI haemorrhage, Cortisol, Dehydration, CHF Renal), Renal Failure, Post Renal (Malignancy, Nephrolithiasis, Prostatism)

Causes of decreased level include Liver disease, SIADH.
CREATININE, SERUM-Higher than normal level may be due to:

Blockage in the urinary tract

- Kidney problems, such as kidney damage or failure, infection, or reduced blood flow
 Loss of body fluid (dehydration)
- 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

Causes of decreased levels-Low Zinc intake, OCP, Multiple Sclerosis

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 globulin

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.

ABO GROUP & RH TYPE, EDTA WHOLE BLOOD-Blood group is identified by antigens and antibodies present in the blood. Antigens are protein molecules found on the surface of red blood cells. Antibodies are found in plasma. To determine blood group, red cells are mixed with different antibody solutions to give A,B,O or AB.

Disclaimer: "Please note, as the results of previous ABO and Rh group (Blood Group) for pregnant women are not available, please check with the patient records for availability of the same."

The test is performed by both forward as well as reverse grouping methods.

THIS REPORT CARRIES THE SIGNATURE OF OUR LABORATORY DIRECTOR. THIS IS AN INVIOLABLE FEATURE OF OUR LAB MANAGEMENT SOFTWARE. HOWEVER, ALL EXAMINATIONS AND INVESTIGATIONS HAVE BEEN CONDUCTED BY OUR PANEL OF DOCTORS.









CLIENT CODE: C000138384 **CLIENT'S NAME AND ADDRESS:**

ACROFEMI HEALTHCARE LTD (MEDIWHEEL)

F-703, LADO SARAI, MEHRAULI SOUTH WEST DELHI

NEW DELHI 110030 DELHI INDIA 8800465156

30-B, CHOWRINGEE MANSION, JAWAHARLAL NEHRU ROAD,

KOLKATA, 700016 WEST BENGAL, INDIA

Tel: 033-22267333,46019048, Fax: 033-22271324

CIN - U74899PB1995PLC045956

PATIENT NAME: DEBABRATA BEHERA

PATIENT ID:

DEBAF150474310

ACCESSION NO:

0082WB00080

48 Years SEX: Male ABHA NO:

REPORTED:

01/03/2023 12:21

DRAWN: 25/02/2023 08:30

RECEIVED: 25/02/2023 08:47

REFERRING DOCTOR: DR. ACROFEMI HEALTHCARE LTD (MEDIWHEEL)

AGE:

CLIENT PATIENT ID:

Test Report Status

<u>Final</u>

Results

Units

MEDI WHEEL FULL BODY HEALTH CHECK UP ABOVE 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. B. N. Jana, MBBS, DCH Consultant

CONDITIONS OF LABORATORY TESTING & REPORTING

- 1. It is presumed that the test sample belongs to the patient named or identified in the test requisition form.
- 2. All tests are performed and reported as per the turnaround time stated in the SRL Directory of Services.
- 3. 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.
- 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 gueries please call customer care (91115 91115) within 48 hours of the report.

SRL Limited

Fortis Hospital, Sector 62, Phase VIII, Mohali 160062



