





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 30-B, CHOWRINGEE MANSION, JAWAHARLAL NEHRU ROAD, KOLKATA, 700016 WEST BENGAL, INDIA Tel : 033-22267333,46019048, Fax : 033-22271324 CIN - U74899PB1995PLC045956

PATIENT NAME : RUSKHSANA KHATOON	PATIENT ID : RUSKF05018482
ACCESSION NO : 0082WB00038 AGE : 39 Years SEX : Female	ABHA NO :
DRAWN : 11/02/2023 10:00 RECEIVED : 11/02/2023 15:14	REPORTED : 13/02/2023 19:46
<b>REFERRING DOCTOR :</b> DR. ACROFEMI HEALTHCARE LTD (MEDIWHEEL)	CLIENT PATIENT ID:

Test Report Status	<u>Final</u>	Results	<b>Biological Reference Interval</b>	Units
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#### MEDI WHEEL FULL BODY HEALTH CHECKUP BELOW 40FEMALE

BLC	DOD COUNTS,EDTA WHOLE BLOOD				
HEN	10GLOBIN (HB)	12.9		12.0 - 15.0	g/dL
ME	THOD : SPECTROPHOTOMETRY				
RED	BLOOD CELL (RBC) COUNT	4.45		3.8 - 4.8	mil/µL
ME	THOD : ELECTRICAL IMPEDANCE				
WH	ITE BLOOD CELL (WBC) COUNT	6.08		4.0 - 10.0	thou/µL
ME	THOD : ELECTRICAL IMPEDANCE				
PLA	TELET COUNT	228		150 - 410	thou/µL
ME	THOD : ELECTRONIC IMPEDENCE & MICROSCOPY				
RB	C AND PLATELET INDICES				
HEN	1ATOCRIT (PCV)	38.2		36 - 46	%
ME	THOD : CALCULATED				
MEA	AN CORPUSCULAR VOLUME (MCV)	85.9		83 - 101	fL
ME	THOD : ELECTRICAL IMPEDANCE				
MEA	AN CORPUSCULAR HEMOGLOBIN (MCH)	29.1		27.0 - 32.0	pg
ME	THOD : CALCULATED				
COI	AN CORPUSCULAR HEMOGLOBIN NCENTRATION (MCHC) ETHOD : CALCULATED	33.9		31.5 - 34.5	g/dL
RED	CELL DISTRIBUTION WIDTH (RDW)	15.3	High	11.6 - 14.0	%
ME	THOD : ELECTRICAL IMPEDANCE				
ME	NTZER INDEX	19.3			
MEA	AN PLATELET VOLUME (MPV)	9.3		6.8 - 10.9	fL
ME	THOD : CALCULATED				
WB	C DIFFERENTIAL COUNT				
NEU	JTROPHILS	58		40 - 80	%
ME	THOD : FLOWCYTOMETRY, ELECTRONIC IMPEDANCE & MICROSCO	DPY.			
LYM	PHOCYTES	31		20 - 40	%
ME	THOD : FLOWCYTOMETRY, ELECTRONIC IMPEDANCE & MICROSCO	)PY.			
MO	NOCYTES	6		2 - 10	%
ME	THOD : FLOWCYTOMETRY, ELECTRONIC IMPEDANCE & MICROSCO	)PY.			
	SINOPHILS	5		1 - 6	%
EOS					
	SOPHILS	0		0 - 2	%
BAS	OPHILS THOD : FLOWCYTOMETRY, ELECTRONIC IMPEDANCE & MICROSCO	-		0 - 2	%
BAS ME		-		0 - 2 2.0 - 7.0	% thou/µL
BAS ME ABS	THOD : FLOWCYTOMETRY, ELECTRONIC IMPEDANCE & MICROSCO	)PY.			











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DRAWN : 11/02/2023 10:00	RECEIVED : 11/02/2023 15:14	REPORTED : 13/02/2023 19:40	6
<b>REFERRING DOCTOR :</b> DR. ACROFEMI	HEALTHCARE LTD ( MEDIWHEEL )	CLIENT PATIENT ID:	
Test Report Status <u>Final</u>	Results	Biological Reference Interva	l Units
ABSOLUTE LYMPHOCYTE COUNT	1.88	1 - 3	thou/µL
METHOD : FLOWCYTOMETRY & CALCULATED			
ABSOLUTE MONOCYTE COUNT	0.36	0.20 - 1.00	thou/µL
METHOD : FLOWCYTOMETRY & CALCULATED			<i>,</i> ,
ABSOLUTE EOSINOPHIL COUNT	0.30	0.02 - 0.50	thou/µL
METHOD : FLOWCYTOMETRY & CALCULATED			
ABSOLUTE BASOPHIL COUNT	0 Low	0.02 - 0.10	thou/µL
METHOD : FLOWCYTOMETRY & CALCULATED			
MORPHOLOGY			
RBC	NORMOCYTIC NORMOCHRO	MIC	
METHOD : MICROSCOPIC EXAMINATION			
WBC	NORMAL MORPHOLOGY		
METHOD : MICROSCOPIC EXAMINATION			
PLATELETS	ADEQUATE		
METHOD : MICROSCOPIC EXAMINATION			
ERYTHROCYTE SEDIMENTATION R BLOOD	ATE (ESR),WHOLE		
E.S.R	5	0 - 20	mm at 1 hr
METHOD : AUTOMATED (PHOTOMETRICAL CAPILL	ARY STOPPED FLOW KINETIC ANALYSIS)"		
GLUCOSE FASTING, FLUORIDE PLA	SMA		
FBS (FASTING BLOOD SUGAR)	77	74 - 100	mg/dL
METHOD : ENZYMATIC (HEXOKINASE/G-6-PDH)			
GLYCOSYLATED HEMOGLOBIN(HB/ BLOOD	A1C), EDTA WHOLE		
HBA1C	5.2	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)	%

< 116.0

METHOD : HPLC ESTIMATED AVERAGE GLUCOSE(EAG) 102.5





mg/dL





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**Biological Reference Interval** Units

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Test Report Status Final

Results

## SRL LIMITED - KOLKATA REF. LAB Bio-Rad Variant II Turbo CDM 5.4 S/N : 13466

Patient Data
Sample ID:
Patient ID:
Name:
Physician:
Sex:
DOB:

8213576421 0082WB000380 RUSKHSANAKHATOON

	Analysis Data
	Analysis Performed:
	Injection Number:
N	Run Number:
	Rack ID:
	Tube Number:
	Report Generated:
	Operator ID:

PATIENT REP V2TURBO\_A1c

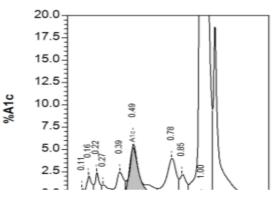
11/02/2023 18:24:37
9044
561
3
11/02/2023 18:42:39
1102202010.42.00

Comments:

Peak Name	NGSP %	Area %	Retention Time (min)	Peak Area
Unknown		0.1	0.110	2051
A1a		0.9	0.157	14260
A1b		0.9	0.216	14539
F		0.6	0.267	9616
LA1c		1.5	0.391	22832
A1c	5.2		0.489	66471
P3		3.4	0.776	52357
P4		1.2	0.854	18656
Ao		87.1	1.000	1358613

Total Area: 1,559,395

### HbA1c (NGSP) = 5.2 %



#### **GLUCOSE, POST-PRANDIAL, PLASMA**











mg/dL

## CLIENT CODE : C000138384

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PPBS(POST PRANDIAL BLOOD SUGAR)

76

140 Normal 140 - 199 Pre-diabetic

> or = 200 Diabetic

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

#### Comments

NOTE: PP SUGAR CAN BE LOWER THAN FASTING SUGAR DUE TO THE FOLLOWING REASONS:

1)OPTIMUM AMOUNT OF GLUCOSE (i.e. 75gm) MAY NOT HAVE BEEN CONSUMED. 2)PATIENT MAY BE A KNOWN DIABETIC UNDER TREATMENT.

3)IN LATENT DIABETICS, HYPERSECRETION OF INSULIN BY THE ISLET CELLS OF PANCREAS MAY LEAD TO INCREASED UTILISATION OF POST PRANDIAL BLOOD GLUCOSE.

4) IN CASE OF HEAVY EXCERCISES LIKE TRADEMILL TEST BEFORE GIVING PP SAMPLE.

5) "DAWN PHENOMENON" WHICH IS HIGH SUGAR VALUE IN THE MORNING DUE TO NORMAL ALTERATION IN HORMONES LIKE GROWTH HORMONE, CORTISOL, EPINEPHRINE AND NOREPINEPHRIN AFTER WAKING UP.

6) TAKING TOO MUCH BLOOD PRESSURE MEDICATION MAY ALSO CAUSE THE BLOOD SUGAR TO GO UP IN THE MORNING.

## LIPID PROFILE, SERUM

CHOLESTEROL, TOTAL	155	< 200 Desirable 200 - 239 Borderline High >/= 240 High	mg/dL
METHOD : ENZYMATIC ASSAY			
TRIGLYCERIDES	80	< 150 Normal 150 - 199 Borderline High 200 - 499 High >/=500 Very High	mg/dL
METHOD : GLYCEROL PHOSPHATE OXIDASE			
HDL CHOLESTEROL	53	Low : < 40 High : > / = 60	mg/dL
METHOD : ACCELERATOR SELECTIVE DETERGENT METHODOLOGY			
CHOLESTEROL LDL	86		mg/dL
NON HDL CHOLESTEROL	102	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	16.0		mg/dL
CHOL/HDL RATIO	2.9		
LDL/HDL RATIO	1.6		









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

#### 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

<b>Risk Category</b>				
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 Hypercholesterolemi	ia		
High Risk	1. Three major ASCVD risk factors. 2. Di	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. 7. Lipoprotein a >/= 50mg/dl 8. 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 premature 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 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)		









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Extreme Risk Group Category B	<or 30<="" =="" th=""><th><or 60<="" =="" th=""><th>&gt; 30</th><th>&gt;60</th></or></th></or>	<or 60<="" =="" th=""><th>&gt; 30</th><th>&gt;60</th></or>	> 30	>60
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	0.49	0.2 - 1.2	mg/dL
METHOD : DIAZONIUM SALT			
BILIRUBIN, DIRECT	0.20	0.0 - 0.5	mg/dL
METHOD : DIAZO REACTION			
BILIRUBIN, INDIRECT	0.29	0.1 - 1.0	mg/dL
METHOD : CALCULATED			
TOTAL PROTEIN	6.9	6.0 - 8.30	g/dL
METHOD : BIURET			
ALBUMIN	4.5	3.5 - 5.2	g/dL
METHOD : COLORIMETRIC (BROMCRESOL GREEN)			
GLOBULIN	2.4	2.0 - 3.5	g/dL
ALBUMIN/GLOBULIN RATIO	1.9	1 - 2.1	RATIO
METHOD : CALCULATED PARAMETER			
ASPARTATE AMINOTRANSFERASE (AST/SGOT)	22	5 - 34	U/L
METHOD : ENZYMATIC (NADH (WITHOUT P-5'-P)			
ALANINE AMINOTRANSFERASE (ALT/SGPT)	23	0 - 55	U/L
METHOD : ENZYMATIC (NADH (WITHOUT P-5'-P)			
ALKALINE PHOSPHATASE	77	40 - 150	U/L
METHOD : PARA-NITROPHENYL PHOSPHATE			
GAMMA GLUTAMYL TRANSFERASE (GGT)	13	8 -33	U/L
METHOD : L-GAMMA-GLUTAMYL-4-NITROANALIDE /GLYCYLGLY0			
LACTATE DEHYDROGENASE	188	125 - 220	U/L
METHOD : IFCC LACTATE TO PYRUVATE			
BLOOD UREA NITROGEN (BUN), SERUM			
BLOOD UREA NITROGEN	10	7.0 - 18.7	mg/dL
METHOD : UREASE METHOD			
CREATININE, SERUM			
CREATININE	0.60	0.50 - 1.00	mg/dL











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Test Report Status	<u>Final</u>	Results		Biological Reference Interv	al Units
METHOD : KINETIC ALKALIN	E PICRATE				
<b>BUN/CREAT RATIO</b>					
BUN/CREAT RATIO		16.67	High	5.0 - 15.0	
URIC ACID, SERUM					
URIC ACID		4.1		2.6 - 6.0	mg/dL
METHOD : URICASE					
TOTAL PROTEIN, SEP	RUM				
TOTAL PROTEIN		6.9		6.0 - 8.3	g/dL
METHOD : BIURET					
ALBUMIN, SERUM					
ALBUMIN		4.5		3.5 - 5.2	g/dL
METHOD : COLORIMETRIC (E	BROMCRESOL GREEN)				
GLOBULIN					
GLOBULIN		2.4		2.0 - 3.5	g/dL
METHOD : CALCULATED PAR	AMETER				
ELECTROLYTES (NA/	K/CL), SERUM				
SODIUM, SERUM		138		136 - 145	mmol/L
METHOD : ION SELECTIVE E	LECTRODE TECHNOLOGY INDIRECT				
POTASSIUM, SERUM		4.50		3.5 - 5.1	mmol/L
METHOD : ION SELECTIVE E	LECTRODE TECHNOLOGY INDIRECT				
CHLORIDE, SERUM		104		98 - 107	mmol/L
METHOD : ION SELECTIVE E	LECTRODE TECHNOLOGY INDIRECT				











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SOUTH WEST DELHI	
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DELHI INDIA	
3800465156	

<u>Final</u>

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Results

## Interpretation(s)

**Test Report Status** 

Sodium	Potassium	Chloride
Decreased in:CCF,cirrhosis, vomiting, diarrhea, excessive sweating, salt-losing nephropathy,adrenal insufficiency, nephrotic syndrome, water intoxication, SIADH. Drugs: thiazides, diuretics, ACE inhibitors, chlorpropamide,carbamazepine,anti depressants (SSRI), antipsychotics.	Decreased in: Low potassium intake,prolonged vomiting or diarrhea, RTA types I and II, hyperaldosteronism, Cushing's syndrome,osmotic diuresis (e.g., hyperglycemia),alkalosis, familial periodic paralysis,trauma (transient).Drugs: Adrenergic agents, diuretics.	Decreased in: Vomiting, diarrhea, renal failure combined with salt deprivation, over-treatment with diuretics, chronic respiratory acidosis, diabetic ketoacidosis, excessive sweating, SIADH, salt-losing nephropathy, porphyria, expansion of extracellular fluid volume, adrenalinsufficiency, hyperaldosteronism,metabolic alkalosis. Drugs: chronic laxative,corticosteroids, diuretics.
Increased in: Dehydration (excessivesweating, severe vomiting or diarrhea),diabetes mellitus, diabetesinsipidus, hyperaldosteronism, inadequate water intake. Drugs: steroids, licorice,oral contraceptives.	Increased in: Massive hemolysis, severe tissue damage, rhabdomyolysis, acidosis, dehydration,renal failure, Addison's disease, RTA type IV, hyperkalemic familial periodic paralysis. Drugs: potassium salts, potassium- sparing diuretics,NSAIDs, beta-blockers, ACE inhibitors, high- dose trimethoprim-sulfamethoxazole.	Increased in: Renal failure, nephrotic syndrome, RTA, dehydration, overtreatment with saline, hyperparathyroidism, diabetes insipidus, metabolic acidosis from diarrhea (Loss of HCO3-), respiratory alkalosis, hyperadrenocorticism. Drugs: acetazolamide, androgens, hydrochlorothiazide, salicylates.
Interferences: Severe lipemia or hyperproteinemi, if sodium analysis involves a dilution step can cause spurious results. The serum sodium falls about 1.6 mEq/L for each 100 mg/dL increase in blood glucose.	Interferences: Hemolysis of sample, delayed separation of serum, prolonged fist clenching during blood drawing, and prolonged tourniquet placement. Very high WBC/PLT counts may cause spurious. Plasma potassium levels are normal.	Interferences:Test is helpful in assessing normal and increased anion gap metabolic acidosis and in distinguishing hypercalcemia due to hyperparathyroidism (high serum chloride) from that due to malignancy (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
METHOD : DIPSTICK		











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DRAWN: 11/02/2023 10:00	RECEIVED : 11/02/2023 15:14	REPORTED : 13/02/2023 19:46
REFERRING DOCTOR : DR. ACROFE	MI HEALTHCARE LTD ( MEDIWHEEL )	CLIENT PATIENT ID :

REFERRING DOCTOR : DR. ACROFEMI HEALTHCARE LTD ( MEDIWHEEL )

Test Report Status <u>Final</u>	Results	Biological Reference Interva	al Units
UROBILINOGEN	NORMAL	NORMAL	
METHOD : DIPSTICK			
NITRITE	NOT DETECTED	NOT DETECTED	
METHOD : DIPSTICK			
LEUKOCYTE ESTERASE	NEGATIVE	NOT DETECTED	
MICROSCOPIC EXAMINATION, URINE			
RED BLOOD CELLS	NOT DETECTED	NOT DETECTED	/HPF
PUS CELL (WBC'S)	1-2	0-5	/HPF
EPITHELIAL CELLS	1-2	0-5	/HPF
CASTS	NOT DETECTED		
CRYSTALS	NOT DETECTED		
BACTERIA	NOT DETECTED	NOT DETECTED	
YEAST	NOT DETECTED	NOT DETECTED	

#### Comments

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











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CIN - U74899PB1995PLC045956

PATIENT NAME : RUSKHSANA KI	HATOON	PATIENT ID : RUSKF05018482
ACCESSION NO : 0082WB00038	AGE : 39 Years SEX : Female	ABHA NO :
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<b>REFERRING DOCTOR :</b> DR. ACROFEM	II HEALTHCARE LTD ( MEDIWHEEL )	CLIENT PATIENT ID:
Test Report Status Final	Results	Biological Reference Interval Units

### 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

ТЗ	117.7	35 - 193
METHOD : TWO-STEP CHEMILUMINESCENT MICROPARTICLE IM	IUNOASSAY	
T4	8.07	Non-Pregnant Women 4.87 - 11.71

METHOD : TWO-STEP CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY TSH (ULTRASENSITIVE) 2.459

Pregnant Women

0.350 - 4.940

1st Trimester: 7.33 - 14.80 2nd Trimester: 7.93 - 16.10 3rd Trimester: 6.95 - 15.70

µIU/mL

ng/dL

µg/dL









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Test Report Status Final	Results	Biological Reference Interval Units

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METHOD : TWO-STEP CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY

#### 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.

### PAPANICOLAOU SMEAR

TEST METHOD

SPECIMEN TYPE

### PHYSICAL EXAMINATION, STOOL

CONSISTENCY

METHOD : MANUAL

SAMPLE NOT RECEIVED SAMPLE NOT RECEIVED

SAMPLE NOT RECEIVED











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PATIENT NAME : RUSKHSANA K	PATIENT ID : RUSKF05018482	
ACCESSION NO : 0082WB00038	AGE : 39 Years SEX : Female	ABHA NO :
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Test Report Status <u>Final</u>	Results	Biological Reference Interval Units
ABO GROUP & RH TYPE, EDTA WHOLE	BLOOD	
ABO GROUP	TYPE O	
METHOD : GEL CARD METHOD		
RH TYPE	NEGATIVE	
METHOD : GEL CARD METHOD		
XRAY-CHEST		
IMPRESSION	zone.	ds are show prominent vascular marking in both lower of diaphragm appear flattened.
TMT OR ECHO		
TMT OR ECHO	ECHO DONE INSTE	
ECG		
ECG	WITHIN NORMAL L	IMITS
MEDICAL HISTORY		
RELEVANT PRESENT HISTORY	NOT SIGNIFICANT	
RELEVANT PAST HISTORY	NOT SIGNIFICANT	
RELEVANT PERSONAL HISTORY	NOT SIGNIFICANT	
RELEVANT FAMILY HISTORY	MOTHER : BRONCH	IIAL ASTHMA
OCCUPATIONAL HISTORY	NOT SIGNIFICANT	
HISTORY OF MEDICATIONS	NOT SIGNIFICANT	
ANTHROPOMETRIC DATA & BMI		
HEIGHT IN METERS	1.61	mts
WEIGHT IN KGS.	77	Kgs
ВМІ	30	BMI & Weight Status as follows: kg/sqmts Below 18.5: Underweight 18.5 - 24.9: Normal 25.0 - 29.9: Overweight

### **GENERAL EXAMINATION**

MENTAL / EMOTIONAL STATE	NORMAL
PHYSICAL ATTITUDE	NORMAL
GENERAL APPEARANCE / NUTRITIONAL STATUS	OBESE
BUILT / SKELETAL FRAMEWORK	AVERAGE
FACIAL APPEARANCE	NORMAL
SKIN	NORMAL



30.0 and Above: Obese





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PATIENT NAME: RUSKHSANA KHATOON	PATIENT ID : RUSKF05018482
ACCESSION NO : 0082WB00038 AGE : 39 Years SEX : Female	ABHA NO :
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Test Report Status <u>Final</u>	Results	Biological Reference Interval Units
UPPER LIMB	NORMAL	
LOWER LIMB	NORMAL	
NECK	NORMAL	
NECK LYMPHATICS / SALIVARY GLANDS	NOT ENLARGED OR TENDE	-R
THYROID GLAND	NOT ENLARGED	
	NORMAL	
TEMPERATURE	NORMAL	
PULSE	87/MINS	
RESPIRATORY RATE	NORMAL	
CARDIOVASCULAR SYSTEM		
BP	118/72	mm/Hg
PERICARDIUM	NORMAL	
APEX BEAT	NORMAL	
HEART SOUNDS	S1, S2 HEARD NORMALLY	
MURMURS	ABSENT	
RESPIRATORY SYSTEM		
SIZE AND SHAPE OF CHEST	NORMAL	
MOVEMENTS OF CHEST	SYMMETRICAL	
BREATH SOUNDS INTENSITY	NORMAL	
BREATH SOUNDS QUALITY	VESICULAR (NORMAL)	
ADDED SOUNDS	ABSENT	
PER ABDOMEN		
APPEARANCE	NORMAL	
VENOUS PROMINENCE	ABSENT	
LIVER	NOT PALPABLE	
SPLEEN	NOT PALPABLE	
CENTRAL NERVOUS SYSTEM		
HIGHER FUNCTIONS	NORMAL	
CRANIAL NERVES	NORMAL	
CEREBELLAR FUNCTIONS	NORMAL	
SENSORY SYSTEM	NORMAL	
MOTOR SYSTEM	NORMAL	
REFLEXES	NORMAL	
MUSCULOSKELETAL SYSTEM		











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PATIENT NAME: RUSKHSANA KHATOON	PATIENT ID : RUSKF05018482
ACCESSION NO : 0082WB00038 AGE : 39 Years SEX : Female	ABHA NO :
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Test Report Status <u>Final</u>	Results	Biological Reference Interval Units	
SPINE	NORMAL		
JOINTS	NORMAL		
BASIC EYE EXAMINATION			
CONJUNCTIVA	NORMAL		
EYELIDS	NORMAL		
EYE MOVEMENTS	NORMAL		
DISTANT VISION RIGHT EYE WITHOUT GLASSES	6/6		
DISTANT VISION LEFT EYE WITHOUT GLASSES	6/6		
NEAR VISION RIGHT EYE WITHOUT GLASSES	N6		
NEAR VISION LEFT EYE WITHOUT GLASSES	N6		
COLOUR VISION	NORMAL		
BASIC ENT EXAMINATION			
EXTERNAL EAR CANAL	NORMAL		
TYMPANIC MEMBRANE	NORMAL		
NOSE	NO ABNORMALITY DETECT	ED	
SINUSES	NORMAL		
THROAT	NO ABNORMALITY DETECT	ED	
TONSILS	NOT ENLARGED		
BASIC DENTAL EXAMINATION			
TEETH	NORMAL		
GUMS	HEALTHY		
SUMMARY			
REMARKS / RECOMMENDATIONS	OVERWEIGHT (77 kgs). & RATIO(16.67) ADVISED : 1. DIET MODIFICATION AS 2. REDUCE BODY WEIGHT KGS). 3. REGULAR PHYSICAL EXI 4. DRINK PLENTY OF WATE	(ESTIMATED BODY WEIGHT SHOULD BE : 61 ERCISE AND WALKING.	L

#### Comments

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











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#### 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. 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 text 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,

#### salicvlates)

**REFERENCE** :

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

#### Increased in

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

Decreased in

Pancreatic islet cell disease with increased insulin, insulinoma, adrenocortical insufficiency, hypopituitarism, diffuse liver disease, malignancy (adrenocortical,

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 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



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PATIENT NAME : RUSKHSANA K	HATOON	PATIENT ID : RUSKF05018482

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#### 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

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, hereditary and neonatal jaundice). Conjugated (direct) bilirubin is elevated more than unconjugated (indirect) bilirubin in Viral hepatitis, Drug reactions, Alcoholic liver disease Conjugated (direct) bilirubin is also elevated more than unconjugated (indirect) bilirubin when there is some kind of blockage of the bile ducts like in Gallstones getting into the bile ducts, tumors &Scarring of the bile ducts. Increased unconjugated (indirect) bilirubin may be a result of Hemolytic or pernicious anemia, Transfusion reaction & a common metabolic condition termed Gilbert syndrome, due to low levels of the enzyme that attaches sugar molecules to bilirubin.

AST is an enzyme found in various parts of the body. AST is found in the liver, heart, skeletal muscle, kidneys, brain, and red blood cells, and it is commonly measured clinically as a marker for liver health. AST levels increase during chronic viral hepatitis, blockage of the bile duct, cirrhosis of the liver, liver cancer, kidney failure, hemolytic anemia, pancreatitis, hemochromatosis. AST levels may also increase after a heart attack or strenuous activity. ALT test measures the amount of this enzyme in the blood. ALT is found mainly in the liver, but also in smaller amounts in the kidneys, heart, muscles, and pancreas. It is commonly measured as a part of a diagnostic evaluation of hepatocellular injury, to determine liver health.AST levels increase during acute hepatitis, sometimes due to a viral infection, ischemia to the liver, chronic hepatitis, obstruction of bile ducts, cirrhosis.

ALP is a protein found in almost all body tissues. Tissues with higher amounts of ALP include the liver, bile ducts and bone. Elevated ALP levels are seen in Biliary obstruction, Osteoblastic bone tumors, osteomalacia, hepatitis, Hyperparathyroidism, Leukemia, Lymphoma, Paget'''''''s disease, Rickets, Sarcoidosis etc. Lower-than-normal ALP levels seen in Hypophosphatasia, Malnutrition, Protein deficiency, Wilson'''''''s disease. GGT is an enzyme found in cell membranes of many tissues mainly in the liver, kidney and pancreas. It is also found in other tissues including intestine, 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

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:

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, 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

Higher-than-normal levels may be due to: Chronic inflammation or infection, including HIV and hepatitis B or C, Multiple myeloma, Waldenstrom" 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.











8800465156

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

SRL Ltd 30-B, CHOWRINGEE MANSION, JAWAHARLAL NEHRU ROAD, KOLKATA, 700016 WEST BENGAL, INDIA Tel : 033-22267333,46019048, Fax : 033-22271324 CIN - U74899PB1995PLC045956

Test Report Status <u>Final</u>	Results	Biological Reference Interval Units
REFERRING DOCTOR : DR. ACROFEM	II HEALTHCARE LTD ( MEDIWHEEL )	CLIENT PATIENT ID :
DRAWN : 11/02/2023 10:00	RECEIVED : 11/02/2023 15:14	REPORTED : 13/02/2023 19:46
ACCESSION NO : 0082WB00038	AGE : 39 Years SEX : Female	ABHA NO :
PATIENT NAME : RUSKHSANA K	HATOON	PATIENT ID : RUSKF05018482

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

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











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REFERRING DOCT	OR: DR. ACROFEM	HEALTHCARE LTD ( N	MEDIWHEEL )	CLIENT	PATIENT ID	:
ſ						

Test Report Status Final

Results

Units

#### MEDI WHEEL FULL BODY HEALTH CHECKUP BELOW 40FEMALE

ULTRASOUND ABDOMEN

ULTRASOUND ABDOMEN

<u>IMPRESSION</u>: No significant abnormality.

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

Dr. B. N. Jana, MBBS, DCH Consultant

CONDITIONS OF LABORAT	ORY TESTING & REPORTING
<ol> <li>It is presumed that the test sample belongs to the patient named or identified in the test requisition form.</li> <li>All tests are performed and reported as per the turnaround time stated in the SRL Directory of Services.</li> <li>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.</li> <li>A requested test might not be performed if:         <ol> <li>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</li> </ol> </li> </ol>	<ol> <li>SRL confirms that all tests have been performed or assayed with highest quality standards, clinical safety &amp; technical integrity.</li> <li>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.</li> <li>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.</li> <li>Test results cannot be used for Medico legal purposes.</li> <li>In case of queries please call customer care (91115 91115) within 48 hours of the report.</li> </ol>
	SRL Limited





