

CODE/NAME & ADDRESS: C000049066 SRL JAIPUR WELLNESS CORPORATE WALK IN AAKRITI LABS PVT LTD. A-430, AGRASEN MARG

**JAIPUR 302017** 9314660100

ACCESSION NO: 0251WF000685 PATIENT ID : HARSF100695251

CLIENT PATIENT ID: 012306100047

ABHA NO

AGE/SEX :28 Years Female :10/06/2023 10:24:00 DRAWN RECEIVED: 10/06/2023 11:30:04

REPORTED :10/06/2023 16:14:11

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

	AEMATOLOGY - CBC		
MEDI WHEEL FULL BODY HEALTH CHECKUP BE	LOW 40FEMALE		
BLOOD COUNTS,EDTA WHOLE BLOOD			
HEMOGLOBIN (HB)  METHOD: CYANIDE FREE DETERMINATION	12.8	12.0 - 15.0	g/dL
RED BLOOD CELL (RBC) COUNT METHOD: ELECTRICAL IMPEDANCE	5.00 High	3.8 - 4.8	mi <b>l</b> /μL
WHITE BLOOD CELL (WBC) COUNT METHOD: ELECTRICAL IMPEDANCE	6.60	4.0 - 10.0	thou/µL
PLATELET COUNT  METHOD: ELECTRONIC IMPEDANCE	244	150 - 410	thou/μL
RBC AND PLATELET INDICES			
HEMATOCRIT (PCV)	38.9	36 <b>-</b> 46	%
METHOD: CALCULATED PARAMETER  MEAN CORPUSCULAR VOLUME (MCV)  METHOD: CALCULATED PARAMETER	78.0 Low	83 - 101	fL
MEAN CORPUSCULAR HEMOGLOBIN (MCH) METHOD: CALCULATED PARAMETER	25.6 Low	27.0 - 32.0	pg
MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION (MCHC) METHOD: CALCULATED PARAMETER	32.9	31.5 - 34.5	g/dL
RED CELL DISTRIBUTION WIDTH (RDW) METHOD: CALCULATED PARAMETER	15.4 High	11.6 - 14.0	%
MENTZER INDEX	15.6		
MEAN PLATELET VOLUME (MPV)  METHOD: CALCULATED PARAMETER	10.2	6.8 - 10.9	fL
WBC DIFFERENTIAL COUNT			
NEUTROPHILS  METHOD: IMPEDANCE WITH HYDRO FOCUS AND MICROSCOPY	48	40 - 80	%
LYMPHOCYTES  METHOD: IMPEDANCE WITH HYDRO FOCUS AND MICROSCOPY	45 High	20 - 40	%
MONOCYTES	05	2 - 10	%

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METHOD: IMPEDANCE WITH HYDRO FOCUS AND MICROSCOPY			0.4
EOSINOPHILS	02	1 - 6	%
METHOD: IMPEDANCE WITH HYDRO FOCUS AND MICROSCOPY			
BASOPHILS	00	0 - 2	%
METHOD: IMPEDANCE WITH HYDRO FOCUS AND MICROSCOPY			
ABSOLUTE NEUTROPHIL COUNT	3.17	2.0 - 7.0	thou/µL
METHOD: CALCULATED PARAMETER			
ABSOLUTE LYMPHOCYTE COUNT	2.97	1.0 - 3.0	thou/µL
METHOD: CALCULATED PARAMETER			
ABSOLUTE MONOCYTE COUNT	0.33	0.2 - 1.0	thou/µL
METHOD: CALCULATED PARAMETER			
ABSOLUTE EOSINOPHIL COUNT	0.13	0.02 - 0.50	thou/µL
METHOD: CALCULATED PARAMETER			•
ABSOLUTE BASOPHIL COUNT	0 Low	0.02 - 0.10	thou/µL
		3.32 3113	
NEUTROPHIL LYMPHOCYTE RATIO (NLR)	1.1		

#### 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.

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.

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.

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**PATIENT NAME: HARSHITA AGARWAL** 

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ACCESSION NO: 0251WF000685 AGE

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# **HAEMATOLOGY**

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

# ERYTHROCYTE SEDIMENTATION RATE (ESR), WHOLE BLOOD

E.S.R **29 High** 0 - 20 mm at 1 hr

METHOD: AUTOMATED (PHOTOMETRICAL CAPILLARY STOPPED FLOW KINETIC ANALYSIS)"

#### Interpretation(s)

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)

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, salividaes)

#### 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."

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# **IMMUNOHAEMATOLOGY**

## MEDI WHEEL FULL BODY HEALTH CHECKUP BELOW 40FEMALE

# **ABO GROUP & RH TYPE, EDTA WHOLE BLOOD**

ABO GROUP TYPE B

METHOD: TUBE AGGLUTINATION

RH TYPE POSITIVE

METHOD: TUBE AGGLUTINATION

Interpretation(s)

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

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

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

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mg/dL

%

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

**BIOCHEMISTRY** 

MEDI WHEEL FULL BODY HEALTH CHECKUP BELOW 40FEMALE

**GLUCOSE FASTING, FLUORIDE PLASMA** 

FBS (FASTING BLOOD SUGAR) 74 - 99 94

METHOD: GLUCOSE OXIDASE

GLYCOSYLATED HEMOGLOBIN(HBA1C), EDTA WHOLE **BLOOD** 

HBA1C 5.6 Non-diabetic: < 5.7

Pre-diabetics: 5.7 - 6.4 Diabetics: > or = 6.5Therapeutic goals: < 7.0 Action suggested: > 8.0 (ADA Guideline 2021)

METHOD: HIGH PERFORMANCE LIQUID CHROMATOGRAPHY (HPLC)

mg/dL ESTIMATED AVERAGE GLUCOSE(EAG) 114.0 < 116.0

METHOD: CALCULATED PARAMETER

**GLUCOSE, POST-PRANDIAL, PLASMA** 

PPBS(POST PRANDIAL BLOOD SUGAR) 70 - 140 99 mg/dL

METHOD: GLUCOSE OXIDASE

LIPID PROFILE, SERUM

METHOD: CHOLESTEROL OXIDASE

CHOLESTEROL, TOTAL 173 mg/dL < 200 Desirable

200 - 239 Borderline High

>/= 240 High

TRIGLYCERIDES 117 < 150 Normal mg/dL

150 - 199 Borderline High

200 - 499 High >/=500 Very High

METHOD: LIPASE/GPO-PAP NO CORRECTION

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HDL CHOLESTEROL	41	< 40 Low mg/dL >/=60 High
METHOD: DIRECT CLEARANCE METHOD		
CHOLESTEROL LDL	109 High	< 100 Optimal mg/dL 100 - 129 Near optimal/ above optimal
		130 - 159 Borderline High 160 - 189 High
		>/= 190 Very High
NON HDL CHOLESTEROL	132 High	Desirable: Less than 130 mg/dL Above Desirable: 130 - 159 Borderline High: 160 - 189 High: 190 - 219 Very high: > or = 220
METHOD: CALCULATED PARAMETER		, 3
VERY LOW DENSITY LIPOPROTEIN	23.4	=30.0</math mg/dL
CHOL/HDL RATIO	4.2	3.3 - 4.4  Low Risk 4.5 - 7.0  Average Risk 7.1 - 11.0  Moderate Risk > 11.0
LDL/HDL RATIO	2.7	High Risk 0.5 - 3.0 Desirable/Low Risk 3.1 - 6.0 Borderline/Moderate Risk >6.0 High Risk

## Interpretation(s)

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

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Risk Category	
Extreme risk group	A.CAD with > 1 feature of high risk group
_	B. CAD with > 1 feature of Very high risk group or recurrent ACS (within 1 year) despite LDL-C < or =
	50 mg/dl or polyvascular disease







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# Test Report Status Final

#### Results

# Biological Reference Interval Units

Very High Risk	1. Established ASCVD 2. Diabetes with 2 major risk factors or evidence of end organ damage 3.		
	Familial Homozygous Hypercholesterolemi	Familial Homozygous Hypercholesterolemia	
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 >1	90 mg/dl 5. Extreme of a single risk factor. 6. Coronary	
	Artery Calcium - CAC >300 AU. 7. Lipopr	rotein 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 (Ath	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		3. Current Cigarette smoking or tobacco use	
2. Family history of p	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 Therap		Treatment Goals		herapy
	LDL-C (mg/dl)	Non-HDL (mg/dl)	LDL-C (mg/dl)	Non-HDL (mg/dl)
Extreme Risk Group Category A	<50 (Optional goal	< 80 (Optional goal	>OR = 50	>OR = 80
	< OR = 30 )	<OR = 60)		
Extreme Risk Group Category B	<OR = 30	<OR = 60	> 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

<sup>\*</sup>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	0.50	0 - 1	mg/dL
METHOD: DIAZO WITH SULPHANILIC ACID BILIRUBIN, DIRECT	0.17	0.00 - 0.25	mg/dL
METHOD: DIAZO WITH SULPHANILIC ACID BILIRUBIN, INDIRECT	0.33	0.1 - 1.0	mg/dL
METHOD : CALCULATED PARAMETER TOTAL PROTEIN	8.3 High	6.4 - 8.2	g/dL
METHOD: BIURET REACTION, END POINT ALBUMIN	4.5 High	3.8 - 4.4	g/dL
METHOD: BROMOCRESOL GREEN GLOBULIN	3.8	2.0 - 4.1	g/dL
METHOD : CALCULATED PARAMETER ALBUMIN/GLOBULIN RATIO	1.2	1.0 - 2.1	RATIO
METHOD: CALCULATED PARAMETER  ASPARTATE AMINOTRANSFERASE(AST/SGOT)  METHOD: TRIS BUFFER NO P5P IFCC / SFBC 37° C	26	0 - 31	U/L



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ALANINE AMINOTRANSFERASE (ALT/SGPT)  METHOD: TRIS BUFFER NO P5P IFCC / SFBC 37° C	28	0 - 31	U/L
ALKALINE PHOSPHATASE  METHOD: AMP OPTIMISED TO IFCC 37° C	83	39 - 117	U/L
GAMMA GLUTAMYL TRANSFERASE (GGT)  METHOD: GAMMA GLUTAMYL-3 CARBOXY-4 NITROANILIDE (IFCC	16	7 - 32	U/L
LACTATE DEHYDROGENASE	278	230 - 460	U/L
BLOOD UREA NITROGEN (BUN), SERUM			
BLOOD UREA NITROGEN METHOD: UREASE KINETIC	7	5.0 - 18.0	mg/dL
CREATININE, SERUM			
CREATININE  METHOD: ALKALINE PICRATE NO DEPROTEINIZATION	0.76	0.6 - 1.2	mg/dL
BUN/CREAT RATIO			
BUN/CREAT RATIO  METHOD: CALCULATED PARAMETER	9.21		
URIC ACID, SERUM			
URIC ACID  METHOD: URICASE PEROXIDASE WITH ASCORBATE OXIDASE	6.1 High	2.4 - 5.7	mg/dL
TOTAL PROTEIN, SERUM			
TOTAL PROTEIN  METHOD: BIURET REACTION, END POINT	8.3	6.4 - 8.3	g/dL

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**ALBUMIN, SERUM** 

ALBUMIN 4.5 High 3.8 - 4.4 g/dL METHOD: BROMOCRESOL GREEN

**GLOBULIN** 

GLOBULIN 3.8 2.0 - 4.1 g/dL

**ELECTROLYTES (NA/K/CL), SERUM** 

SODIUM, SERUM

METHOD: ION-SELECTIVE ELECTRODE

POTASSIUM, SERUM

METHOD: ION-SELECTIVE ELECTRODE

CHLORIDE, SERUM

142.9

137 - 145

mmol/L

mmol/L

98 - 107

mmol/L

METHOD: ION-SELECTIVE ELECTRODE

#### 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, chlorpropamide,carbamazepine,anti	periodic paralysis,trauma (transient).Drugs: Adrenergic agents,	nephropathy, porphyria, expansion of extracellular fluid volume,
depressants (SSRI), antipsychotics.	diuretics.	adrenalinsufficiency,
		hyperaldosteronism, metabolic
		alkalosis. Drugs: chronic
		laxative,corticosteroids, diuretics.

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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, or al contraceptives. potassium- sparing diuretics, NSAIDs, alkalosis, hyperadre no corticis m 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 may cause spurious. Plasma potassium mg/dL increase in blood glucose. chloride) from that due to malignancy (Normal serum chloride) levels are normal.

# Interpretation(s)

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.
- Diagnosing diabetes.
   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 :

- 1. 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 2. Vitamin C & E are reported to falsely lower test results. (possibly by inhibiting glycation of hemoglobin.
- 3. 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.
- 4. Interference of hemoglobinopathies in HbA1c estimation is seen in
- a) Homozygous hemoglobinopathy. Fructosamine is recommended for testing of HbA1c.
- b) Heterozygous state detected (Ó10 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, 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
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







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MC-5333

**REF. DOCTOR:** SELF **PATIENT NAME: HARSHITA AGARWAL** 

CODE/NAME & ADDRESS: C000049066 SRL JAIPUR WELLNESS CORPORATE WALK IN AAKRITI LABS PVT LTD. A-430, AGRASEN MARG

1ATPUR 302017 9314660100

ACCESSION NO: 0251WF000685 PATIENT ID : HARSF100695251

CLIENT PATIENT ID: 012306100047

ABHA NO

AGE/SEX :28 Years Female DRAWN :10/06/2023 10:24:00 RECEIVED: 10/06/2023 11:30:04

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**Test Report Status** Results **Biological Reference Interval** Units **Final** 

(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, Pagets disease, Rickets, Sarcoidosis etc. Lower-than-normal ALP levels seen in Hypophosphatasia, Malnutrition, Protein deficiency, Wilsons 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, billiary system and pancreas. Conditions that increase serum GGT are obstructive liver disease, high alcohol consumption and use of enzyme-inducing drugs etc.

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 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 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, Muscuophy

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-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, Waldenstroms disease. Lower-than-normal levels may be due to: Agammaglobulinemia, Bleeding (hemorrhage), Burns, Glomerulonephritis, Liver disease, Malabsorption, Malnutrition, Nephrotic syndrome,Protein-losing enteropathy etc. ALBUMIN, SERUM-

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

Dr. Akansha Jain **Consultant Pathologist** 



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CODE/NAME & ADDRESS: C000049066 SRL JAIPUR WELLNESS CORPORATE WALK IN AAKRITI LABS PVT LTD. A-430, AGRASEN MARG

**JAIPUR 302017** 9314660100

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

#### **CLINICAL PATH - URINALYSIS**

# MEDI WHEEL FULL BODY HEALTH CHECKUP BELOW 40FEMALE

PHYSICAL EXAMINATION, URINE

PALE YELLOW **COLOR** 

METHOD: GROSS EXAMINATION

**CLEAR** APPEARANCE

METHOD: GROSS EXAMINATION

#### **CHEMICAL EXAMINATION, URINE**

PH	6 <b>.</b> 5	4.7 - 7.5

METHOD: DOUBLE INDICATOR PRINCIPLE

SPECIFIC GRAVITY 1.015 1.003 - 1.035

METHOD: IONIC CONCENTRATION METHOD PROTFIN **NOT DETECTED** 

**NEGATIVE** METHOD: PROTEIN ERROR OF INDICATORS WITH REFLECTANCE

**NOT DETECTED NEGATIVE** GLUCOSE

METHOD: GLUCOSE OXIDASE PEROXIDASE / BENEDICTS

**NOT DETECTED KETONES NOT DETECTED** 

METHOD: SODIUM NITROPRUSSIDE REACTION **BLOOD** NOT DETECTED **NEGATIVE** 

METHOD: PEROCIDASE ANTI PEROXIDASE

**NOT DETECTED NOT DETECTED BILIRUBIN** 

METHOD : DIPSTICK

**UROBILINOGEN NORMAL** NORMAL

**NOT DETECTED NOT DETECTED** NITRITE

METHOD: NITRATE TO NITRITE CONVERSION METHOD LEUKOCYTE ESTERASE NOT DETECTED **NOT DETECTED** 

MICROSCOPIC EXAMINATION, URINE

METHOD: EHRLICH REACTION REFLECTANCE

/HPF RED BLOOD CELLS **NOT DETECTED NOT DETECTED** 

METHOD: MICROSCOPIC EXAMINATION /HPF PUS CELL (WBC'S) 1-2 0-5

METHOD: DIPSTICK, MICROSCOPY

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	<b>;</b>			
Test Report Status <u>Final</u>	Results	Biological Reference 1	Interval Units	
EPITHELIAL CELLS	2 <b>-</b> 3	0 <b>-</b> 5	/HPF	
METHOD: MICROSCOPIC EXAMINATION				
CASTS	NOT DETECTED			
METHOD: MICROSCOPIC EXAMINATION				
CRYSTALS	NOT DETECTED			
METHOD: MICROSCOPIC EXAMINATION				
BACTERIA	NOT DETECTED	NOT DETECTED		
METHOD: MICROSCOPIC EXAMINATION				
YEAST	NOT DETECTED	NOT DETECTED		

# 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		
	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	

Dr. Akansha Jain **Consultant Pathologist** 





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SRL JAIPUR WELLNESS CORPORATE WALK IN
AAKRITI LABS PVT LTD. A-430, AGRASEN MARG

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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

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**CYTOLOGY** 

## MEDI WHEEL FULL BODY HEALTH CHECKUP BELOW 40FEMALE

**PAPANICOLAOU SMEAR** 

TEST METHOD

SAMPLE NOT RECEIVED

Dr. Akansha Jain Consultant Pathologist





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**REF. DOCTOR:** SELF

MC-5333

PATIENT NAME: HARSHITA AGARWAL

CODE/NAME & ADDRESS: C000049066

SRL JAIPUR WELLNESS CORPORATE WALK IN
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# **CLINICAL PATH - STOOL ANALYSIS**

# MEDI WHEEL FULL BODY HEALTH CHECKUP BELOW 40FEMALE PHYSICAL EXAMINATION, STOOL

COLOUR SAMPLE NOT RECEIVED

METHOD : GROSS EXAMINATION

White the species

Dr. Abhishek Sharma Consultant Microbiologist





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#### **SPECIALISED CHEMISTRY - HORMONE**

# MEDI WHEEL FULL BODY HEALTH CHECKUP BELOW 40FEMALE

# THYROID PANEL, SERUM

<b>,</b>			
ТЗ	123.30	60.0 - 181.0	ng/dL
METHOD: CHEMILUMINESCENCE			
T4	10.60	4.5 - 10.9	μg/dL
METHOD: CHEMILUMINESCENCE			
TSH (ULTRASENSITIVE)	2.561	0.550 - 4.780	μIU/mL
METHOD: CHEMILUMINESCENCE			

#### 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







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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 duriing 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.

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

Dr. Akansha Jain Consultant Pathologist





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