

PATIENT NAME : POOJA ASHISH JUNGARE

REF. DOCTOR : SELF

CODE/NAME & ADDRESS :C000138394
ACROFEMI HEALTHCARE LTD (MEDIWHEEL)
F-703, F-703, LADO SARAI, MEHRAULISOUTH- WEST
DELHI
NEW DELHI 110030
8800465156

ACCESSION NO : 0181WC001891
PATIENT ID : POQJF260695181
CLIENT PATIENT ID:
ABHA NO :

AGE/SEX :27 Years Female
DRAWN :
RECEIVED : 30/03/2023 08:17:49
REPORTED :03/04/2023 12:47:25

Test Report Status	Final	Results	Biological Reference Interval	Units
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MEDI WHEEL FULL BODY HEALTH CHECKUP BELOW 40FFEMALE

XRAY-CHEST

IMPRESSION NO ABNORMALITY DETECTED

TMT OR ECHO

TMT OR ECHO NEGATIVE

ECG

ECG WITHIN NORMAL LIMITS

MEDICAL HISTORY

RELEVANT PRESENT HISTORY URI SINCE 3 DAYS.

RELEVANT PAST HISTORY NOT SIGNIFICANT

RELEVANT PERSONAL HISTORY MARRIED / VEG DIET / NO ALLERGIES / NO SMOKING / NO ALCOHOL.

RELEVANT FAMILY HISTORY ASTHMA : MOTHER, GRANDMOTHER.

HISTORY OF MEDICATIONS NOT SIGNIFICANT

ANTHROPOMETRIC DATA & BMI

HEIGHT IN METERS 1.60 mts

WEIGHT IN KGS. 55 Kgs

BMI 21 **BMI & Weight Status as follows** kg/sqmts

Below 18.5: Underweight

18.5 - 24.9: Normal

25.0 - 29.9: Overweight

30.0 and Above: Obese

GENERAL EXAMINATION

MENTAL / EMOTIONAL STATE NORMAL

PHYSICAL ATTITUDE NORMAL

GENERAL APPEARANCE / NUTRITIONAL STATUS HEALTHY

BUILT / SKELETAL FRAMEWORK AVERAGE

FACIAL APPEARANCE NORMAL

SKIN NORMAL

UPPER LIMB NORMAL

LOWER LIMB NORMAL

NECK NORMAL

NECK LYMPHATICS / SALIVARY GLANDS NOT ENLARGED OR TENDER

THYROID GLAND NOT ENLARGED



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S.K. Tower, Hari Niwas, LBS Marg
THANE, 400602
MAHARASHTRA, INDIA
Tel : 9111591115, Fax : CIN - U74899PB1995PLC045956
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CAROTID PULSATION	NORMAL		
TEMPERATURE	NORMAL		
PULSE	82/MIN.REGULAR, ALL PERIPHERAL PULSES WELL FELT, NO CAROTID BRUIT		
RESPIRATORY RATE	NORMAL		
CARDIOVASCULAR SYSTEM			
BP	120/80 MM HG (SUPINE)		mm/Hg
PERICARDIUM	NORMAL		
APEX BEAT	NORMAL		
HEART SOUNDS	NORMAL		
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		
HERNIA	ABSENT		
CENTRAL NERVOUS SYSTEM			
HIGHER FUNCTIONS	NORMAL		
CRANIAL NERVES	NORMAL		
CEREBELLAR FUNCTIONS	NORMAL		
SENSORY SYSTEM	NORMAL		
MOTOR SYSTEM	NORMAL		
REFLEXES	NORMAL		
MUSCULOSKELETAL SYSTEM			
SPINE	NORMAL		



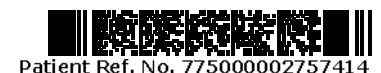
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JOINTS NORMAL
BASIC EYE EXAMINATION
CONJUNCTIVA NORMAL
EYELIDS NORMAL
EYE MOVEMENTS NORMAL
CORNEA NORMAL
DISTANT VISION RIGHT EYE WITHOUT GLASSES REDUCED VISUAL ACUITY 6/9
DISTANT VISION LEFT EYE WITHOUT GLASSES WITHIN NORMAL LIMIT
DISTANT VISION RIGHT EYE WITH GLASSES GLASSES NOT BROUGHT
DISTANT VISION LEFT EYE WITH GLASSES GLASSES NOT BROUGHT
NEAR VISION RIGHT EYE WITHOUT GLASSES WITHIN NORMAL LIMIT
NEAR VISION LEFT EYE WITHOUT GLASSES WITHIN NORMAL LIMIT
COLOUR VISION NORMAL
SUMMARY
RELEVANT HISTORY NOT SIGNIFICANT
RELEVANT GP EXAMINATION FINDINGS NOT SIGNIFICANT
REMARKS / RECOMMENDATIONS LOW FAT, LOW CARBOHYDRATE, HIGH FIBRE DIET.
REGULAR EXERCISE.REGULAR WALK FOR 30-40 MIN DAILY.
REPEAT B.SUGAR,LIPID PROFILE AFTER 3 MONTHS OF DIET AND EXERCISE.
FOLLOW UP WITH GYNAECOLOGIST FOR PCOD.



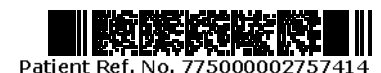
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ULTRASOUND ABDOMEN

ULTRASOUND ABDOMEN

POLYCYSTIC OVARIAN MORPHOLOGY.

Interpretation(s)

MEDICAL

HISTORY-*****

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

****End Of Report****

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HAEMATOLOGY - CBC

MEDI WHEEL FULL BODY HEALTH CHECKUP BELOW 40FEMALE

BLOOD COUNTS,EDTA WHOLE BLOOD

HEMOGLOBIN (HB) METHOD : SLS- HEMOGLOBIN DETECTION METHOD	12.1	12.0 - 15.0	g/dL
RED BLOOD CELL (RBC) COUNT METHOD : HYDRODYNAMIC FOCUSING BY DC DETECTION	4.71	3.8 - 4.8	mil/ μ L
WHITE BLOOD CELL (WBC) COUNT METHOD : FLUORESCENCE FLOW CYTOMETRY	9.08	4.0 - 10.0	thou/ μ L
PLATELET COUNT METHOD : HYDRODYNAMIC FOCUSING BY DC DETECTION	306	150 - 410	thou/ μ L
RBC AND PLATELET INDICES			
HEMATOCRIT (PCV) METHOD : CUMULATIVE PULSE HEIGHT DETECTION METHOD	40.0	36.0 - 46.0	%
MEAN CORPUSCULAR VOLUME (MCV) METHOD : CALCULATED FROM RBC & HCT	84.9	83.0 - 101.0	fL
MEAN CORPUSCULAR HEMOGLOBIN (MCH) METHOD : CALCULATED FROM THE RBC & HGB	25.7 Low	27.0 - 32.0	pg
MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION (MCHC) METHOD : CALCULATED FROM THE HGB & HCT	30.3 Low	31.5 - 34.5	g/dL
RED CELL DISTRIBUTION WIDTH (RDW) METHOD : CALCULATED FROM RBC SIZE DISTRIBUTION CURVE	14.6 High	11.6 - 14.0	%
MENTZER INDEX	18.0		
MEAN PLATELET VOLUME (MPV) METHOD : CALCULATED FROM PLATELET COUNT & PLATELET HEMATOCRIT	11.5 High	6.8 - 10.9	fL
WBC DIFFERENTIAL COUNT			
NEUTROPHILS METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING	54	40 - 80	%
LYMPHOCYTES METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING	34	20 - 40	%
MONOCYTES METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING	5	2 - 10	%
EOSINOPHILS METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING	7 High	1 - 6	%

Priyal Chinchkhede

Dr.Priyal Chinchkhede
Consultant Pathologist

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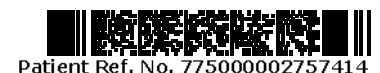
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BASOPHILS METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING	0	0 - 1	%
ABSOLUTE NEUTROPHIL COUNT METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING	4.90	2.0 - 7.0	thou/ μ L
ABSOLUTE LYMPHOCYTE COUNT METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING	3.10 High	1.0 - 3.0	thou/ μ L
ABSOLUTE MONOCYTE COUNT METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING	0.47	0.2 - 1.0	thou/ μ L
ABSOLUTE EOSINOPHIL COUNT METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING	0.63 High	0.02 - 0.50	thou/ μ L
ABSOLUTE BASOPHIL COUNT METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING	0.00 Low	0.02 - 0.10	thou/ μ L
NEUTROPHIL LYMPHOCYTE RATIO (NLR)	1.6		
MORPHOLOGY			
RBC	NORMOCYTIC NORMOCHROMIC		
WBC METHOD : MICROSCOPIC EXAMINATION	NORMAL MORPHOLOGY		
PLATELETS	ADEQUATE		

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 calculatec 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.

Chinchkhede

Dr.Priyal Chinchkhede
 Consultant Pathologist



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HAEMATOLOGY

MEDI WHEEL FULL BODY HEALTH CHECKUP BELOW 40FEMALE

ERYTHROCYTE SEDIMENTATION RATE (ESR),WHOLE BLOOD

E.S.R 6 < 20 mm at 1 hr

METHOD : MODIFIED WESTERGREN

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 automatic 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, Vasculitis, 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 (Paraproteinemia, 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: Polycythemia vera, Sickle cell anemia

LIMITATIONS

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

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

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.

Priyal Chinchkhede

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IMMUNOHAEMATOLOGY

MEDI WHEEL FULL BODY HEALTH CHECKUP BELOW 40 FEMALE

ABO GROUP & RH TYPE, EDTA WHOLE BLOOD

ABO GROUP

TYPE A

RH TYPE

POSITIVE

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.

Chinchkhede

Dr. Priyal Chinchkhede
Consultant Pathologist

Ushma

Dr. Ushma Wartikar
Consultant Pathologist

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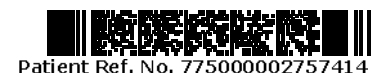
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BIOCHEMISTRY

MEDI WHEEL FULL BODY HEALTH CHECKUP BELOW 40FEMALE

GLUCOSE FASTING,FLUORIDE PLASMA

FBS (FASTING BLOOD SUGAR)	85	Normal 75 - 99	mg/dL
		Pre-diabetics: 100 - 125	
		Diabetic: > or = 126	

METHOD : ENZYMATIC REFERENCE METHOD WITH HEXOKINASE

GLYCOSYLATED HEMOGLOBIN(HBA1C), EDTA WHOLE BLOOD

HBA1C	5.8 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

ESTIMATED AVERAGE GLUCOSE(EAG)	119.8 High	< 116.0	mg/dL
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METHOD : CALCULATED PARAMETER

GLUCOSE, POST-PRANDIAL, PLASMA

PPBS(POST PRANDIAL BLOOD SUGAR)	81	70 - 139	mg/dL
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METHOD : ENZYMATIC REFERENCE METHOD WITH HEXOKINASE

LIPID PROFILE, SERUM

CHOLESTEROL, TOTAL	156	Desirable cholesterol level	mg/dL
		< 200	
		Borderline high cholesterol	
		200 - 239	
		High cholesterol	
		> / = 240	

METHOD : ENZYMATIC COLORIMETRIC ASSAY

TRIGLYCERIDES	68	Normal: < 150	mg/dL
		Borderline high:	
		150 - 199	
		High: 200 - 499	
		Very High: >/= 500	

METHOD : ENZYMATIC COLORIMETRIC ASSAY

HDL CHOLESTEROL	37 Low	Low HDL Cholesterol <40	mg/dL
		High HDL Cholesterol >/= 60	

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Consultant Pathologist

Dr. Priyal Chinchkhede
Consultant Pathologist

Dr. (Mrs) Neelu K Bhojani
Lab Head



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METHOD : ENZYMATIC, COLORIMETRIC

CHOLESTEROL LDL 105 High
 Adult levels: mg/dL
 Optimal < 100
 Near optimal/above optimal:
 100-129
 Borderline high : 130-159
 High : 160-189
 Very high : = 190

METHOD : ENZYMATIC COLORIMETRIC ASSAY

NON HDL CHOLESTEROL 119
 Desirable : < 130 mg/dL
 Above Desirable : 130 -159
 Borderline High : 160 - 189
 High : 190 - 219
 Very high : > / = 220

VERY LOW DENSITY LIPOPROTEIN CHOL/HDL RATIO 13.6
 < OR = 30.0 mg/dL

LDL/HDL RATIO 4.2
 Low Risk : 3.3 - 4.4
 Average Risk : 4.5 - 7.0
 Moderate Risk : 7.1 - 11.0
 High Risk : > 11.0

0.5 - 3.0 Desirable/Low Risk
 3.1 - 6.0 Borderline/Moderate Risk
 >6.0 High Risk

Interpretation(s)

LIVER FUNCTION PROFILE, SERUM

BILIRUBIN, TOTAL 0.34 Upto 1.2 mg/dL
 METHOD : COLORIMETRIC DIAZO

BILIRUBIN, DIRECT 0.18 < 0.30 mg/dL

BILIRUBIN, INDIRECT 0.16 0.1 - 1.0 mg/dL

TOTAL PROTEIN 6.7 6.0 - 8.0 g/dL
 METHOD : COLORIMETRIC

ALBUMIN 4.5 3.97 - 4.94 g/dL
 METHOD : COLORIMETRIC

GLOBULIN 2.2 2.0 - 3.5 g/dL

ALBUMIN/GLOBULIN RATIO 2.1 1.0 - 2.1 RATIO

ASPARTATE AMINOTRANSFERASE (AST/SGOT) 15 < OR = 35 U/L

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MAHARASHTRA, INDIA
Fax :
CIN - U74899PB1995PLC045956



Patient Ref. No. 775000002757414

PATIENT NAME : POOJA ASHISH JUNGARE

REF. DOCTOR : SELF

CODE/NAME & ADDRESS : C000138394
 ACROFEMI HEALTHCARE LTD (MEDIWHEEL)
 F-703, F-703, LADO SARAI, MEHRAULISOUTH- WEST
 DELHI
 NEW DELHI 110030
 8800465156

ACCESSION NO : 0181WC001891
 PATIENT ID : POQJF260695181
 CLIENT PATIENT ID:
 ABHA NO :

AGE/SEX : 27 Years Female
 DRAWN :
 RECEIVED : 30/03/2023 08:17:49
 REPORTED : 03/04/2023 12:47:25

Test Report Status	Final	Results	Biological Reference Interval	Units
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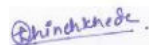
METHOD : UV ABSORBANCE				
ALANINE AMINOTRANSFERASE (ALT/SGPT)	12	< OR = 35		U/L
METHOD : UV ABSORBANCE				
ALKALINE PHOSPHATASE	88	35 - 104		U/L
METHOD : COLORIMETRIC				
GAMMA GLUTAMYL TRANSFERASE (GGT)	9	0 - 40		U/L
METHOD : ENZYMATIC, COLORIMETRIC				
LACTATE DEHYDROGENASE	177	125 - 220		U/L
METHOD : UV ABSORBANCE				
BLOOD UREA NITROGEN (BUN), SERUM				
BLOOD UREA NITROGEN	6	6 - 20		mg/dL
METHOD : ENZYMATIC ASSAY				
CREATININE, SERUM				
CREATININE	0.50	0.5 - 0.9		mg/dL
METHOD : COLORIMETRIC				
BUN/CREAT RATIO				
BUN/CREAT RATIO	12.00	8.0 - 15.0		
URIC ACID, SERUM				
URIC ACID	4.0	2.4 - 5.7		mg/dL
METHOD : ENZYMATIC COLORIMETRIC ASSAY				
TOTAL PROTEIN, SERUM				
TOTAL PROTEIN	6.7	6.0 - 8.0		g/dL
METHOD : COLORIMETRIC				
ALBUMIN, SERUM				
ALBUMIN	4.5	3.97 - 4.94		g/dL
METHOD : COLORIMETRIC				
GLOBULIN				
GLOBULIN	2.2	2.0 - 3.5		g/dL
ELECTROLYTES (NA/K/CL), SERUM				
SODIUM, SERUM	142	136 - 145		mmol/L
POTASSIUM, SERUM	4.08	3.5 - 5.1		mmol/L
CHLORIDE, SERUM	109 High	98 - 107		mmol/L

Interpretation(s)

Sodium	Potassium	Chloride
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Dr. Ushma Wartikar
 Consultant Pathologist



Dr. Priyal Chinchkhede
 Consultant Pathologist



Dr. (Mrs) Neelu K Bhojani
 Lab Head

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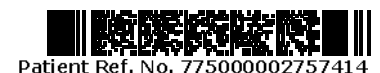
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<p>Decreased in:CCF,cirrhosis, vomiting, diarrhea, excessive sweating, salt-losing nephropathy,adrenal insufficiency, nephrotic syndrome, water intoxication, SIADH. Drugs: thiazides, diuretics, ACE inhibitors, chlorpropamide, carbamazepine, anti depressants (SSRI), anLpsychotics.</p>	<p>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.</p>	<p>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.</p>
<p>Increased in: Dehydration (excessive sweating, severe vomiting or diarrhea),diabetes mellitus, diabetesinsipidus, hyperaldosteronism, inadequate water intake. Drugs: steroids, licorice,oral contraceptives.</p>	<p>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.</p>	<p>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.</p>
<p>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.</p>	<p>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.</p>	<p>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)</p>

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: corbosteroids,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; sulfonlureas, 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 Glycosuria, 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 patient's metabolic control has remained continuously within the target range.
1. eAG (Estimated average glucose) converts percentage HbA1c to mg/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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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 addition 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 (D10 is corrected for HbS & HbC trait.)

c) HbF > 25% on alternate platform (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 Glycosuria, 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 hepatic), 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, 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, biliary 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, 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 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, Muscuopathy

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. Ushma Wartikar
Consultant Pathologist

Dr. Priya Chinchkhede
Consultant Pathologist

Dr. (Mrs) Neelu K Bhojani
Lab Head

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CLINICAL PATH - URINALYSIS

MEDI WHEEL FULL BODY HEALTH CHECKUP BELOW 40FEMALE

PHYSICAL EXAMINATION, URINE

COLOR PALE YELLOW
APPEARANCE CLEAR

CHEMICAL EXAMINATION, URINE

PH 6.0 5.00 - 7.50
SPECIFIC GRAVITY 1.010 1.010 - 1.030

METHOD : URINE ROUTINE & MICROSCOPY EXAMINATION BY INTEGRATED AUTOMATED SYSTEM

PROTEIN NOT DETECTED NOT DETECTED
GLUCOSE NOT DETECTED NOT DETECTED
KETONES NOT DETECTED NOT DETECTED
BLOOD NOT DETECTED NOT DETECTED
UROBILINOGEN NORMAL NORMAL
NITRITE NOT DETECTED NOT DETECTED
LEUKOCYTE ESTERASE NOT DETECTED 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

METHOD : URINE ROUTINE & MICROSCOPY EXAMINATION BY INTEGRATED AUTOMATED SYSTEM

Interpretation(s)

Dr. Priyal Chinchkhede
Consultant Pathologist

Dr. Ushma Wartikar
Consultant Pathologist

Dr. (Mrs) Neelu K Bhojani
Lab Head



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CYTOLOGY

MEDI WHEEL FULL BODY HEALTH CHECKUP BELOW 40FEMALE

PAPANICOLAOU SMEAR

TEST METHOD

SNR

METHOD : MICROSCOPIC EXAMINATION

Chinchkhede

Dr.Priyal Chinchkhede
Consultant Pathologist

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Test Report Status **Final**

Results

Biological Reference Interval Units

CLINICAL PATH - STOOL ANALYSIS

MEDI WHEEL FULL BODY HEALTH CHECKUP BELOW 40FEMALE

PHYSICAL EXAMINATION,STOOL

COLOUR

SAMPLE NOT RECEIVED

METHOD : VISUAL

Sawant

Dr. Sheetal Sawant
Consultant Microbiologist

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SPECIALISED CHEMISTRY - HORMONE

MEDI WHEEL FULL BODY HEALTH CHECKUP BELOW 40 FEMALE

THYROID PANEL, SERUM

T3 **109.0** Non-Pregnant Women **ng/dL**
80.0 - 200.0
Pregnant Women
1st Trimester: 105.0 - 230.0
2nd Trimester: 129.0 - 262.0
3rd Trimester: 135.0 - 262.0

METHOD : ELECTROCHEMILUMINESCENCE

T4 **8.26** Non-Pregnant Women **µg/dL**
5.10 - 14.10
Pregnant Women
1st Trimester: 7.33 - 14.80
2nd Trimester: 7.93 - 16.10
3rd Trimester: 6.95 - 15.70

METHOD : ELECTROCHEMILUMINESCENCE

TSH (ULTRASENSITIVE) **2.190** Non Pregnant Women **µIU/mL**
0.27 - 4.20
Pregnant Women
1st Trimester: 0.33 - 4.59
2nd Trimester: 0.35 - 4.10
3rd Trimester: 0.21 - 3.15

METHOD : ELECTROCHEMILUMINESCENCE

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, Free T4 along with TSH, instead of testing for albumin bound Total T3, Total T4.

Sr. No.	TSH	Total T4	FT4	Total T3	Possible Conditions
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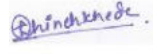
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1	High	Low	Low	Low	(1) Primary Hypothyroidism (2) Chronic autoimmune Thyroiditis (3) Post Thyroidectomy (4) Post Radio-Iodine treatment
2	High	Normal	Normal	Normal	(1)Subclinical Hypothyroidism (2) Patient with insufficient thyroid hormone replacement 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.


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 Consultant Pathologist


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