



CLIENT CODE : C000138383

## 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  
24 SCO, SECTOR 11 D  
CHANDIGARH, 160011  
PUNJAB, INDIA  
Tel : 9111591115, Fax :  
CIN - U74899PB1995PLC045956

PATIENT NAME : NEPAL SINGH

PATIENT ID : NEPAM25086680

ACCESSION NO : 0080VI011002 AGE : 56 Years SEX : Male

ABHA NO :

DRAWN :

RECEIVED : 24/09/2022 09:09

REPORTED : 25/09/2022 09:40

REFERRING DOCTOR : SELF

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Test Report Status	Final	Results	Biological Reference Interval	Units
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**MEDI WHEEL FULL BODY HEALTH CHECK UP ABOVE 40 MALE****BLOOD COUNTS,EDTA WHOLE BLOOD**

HEMOGLOBIN	14.2	13.0 - 17.0	g/dL
RED BLOOD CELL COUNT	5.28	4.5 - 5.5	mil/ $\mu$ L
WHITE BLOOD CELL COUNT	5.00	4.0 - 10.0	thou/ $\mu$ L
PLATELET COUNT	155	150 - 410	thou/ $\mu$ L

**RBC AND PLATELET INDICES**

HEMATOCRIT	43.4	40.0 - 50.0	%
METHOD : ELECTRICAL IMPEDANCE			
MEAN CORPUSCULAR VOL	<b>82.2</b>	<b>Low</b> 83.0 - 101.0	fL
MEAN CORPUSCULAR HGB.	<b>26.9</b>	<b>Low</b> 27.0 - 32.0	pg
MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION	32.7	31.5 - 34.5	g/dL
MENTZER INDEX	15.6		
RED CELL DISTRIBUTION WIDTH	<b>14.4</b>	<b>High</b> 11.6 - 14.0	%
MEAN PLATELET VOLUME	<b>13.1</b>	<b>High</b> 6.8 - 10.9	fL

**WBC DIFFERENTIAL COUNT - NLR**

SEGMENTED NEUTROPHILS	66	40 - 80	%
ABSOLUTE NEUTROPHIL COUNT	3.30	2.0 - 7.0	thou/ $\mu$ L
LYMPHOCYTES	24	20 - 40	%
ABSOLUTE LYMPHOCYTE COUNT	1.20	1.0 - 3.0	thou/ $\mu$ L
NEUTROPHIL LYMPHOCYTE RATIO (NLR)	2.8		
METHOD : CALCULATED PARAMETER			
EOSINOPHILS	2	1 - 6	%
ABSOLUTE EOSINOPHIL COUNT	0.10	0.02 - 0.50	thou/ $\mu$ L
MONOCYTES	8	2 - 10	%
ABSOLUTE MONOCYTE COUNT	0.40	0.2 - 1.0	thou/ $\mu$ L
BASOPHILS	0	0 - 1	%
ABSOLUTE BASOPHIL COUNT	<b>0.00</b>	<b>Low</b> 0.02 - 0.10	thou/ $\mu$ L
METHOD : CALCULATED PARAMETER			

DIFFERENTIAL COUNT PERFORMED ON: AUTOMATED ANALYZER

**ERYTHRO SEDIMENTATION RATE, BLOOD**

SEDIMENTATION RATE (ESR)	<b>30</b>	<b>High</b> 0 - 14	mm at 1 hr
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METHOD : MODIFIED WESTERGREEN

**GLYCOSYLATED HEMOGLOBIN, EDTA WHOLE BLOOD**

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GLYCOSYLATED HEMOGLOBIN (HBA1C)		5.7	Non-diabetic: < 5.7 Pre-diabetics: 5.7 - 6.4 Diabetics: > or = 6.5 ADA Target: 7.0 Action suggested: > 8.0	%
MEAN PLASMA GLUCOSE		<b>116.9</b>	<b>High</b> < 116.0	mg/dL
<b>GLUCOSE, FASTING, PLASMA</b>				
GLUCOSE, FASTING, PLASMA		96	74 - 106	mg/dL
METHOD : HEXOKINASE				
<b>GLUCOSE, POST-PRANDIAL, PLASMA</b>				
GLUCOSE, POST-PRANDIAL, PLASMA		119	Non-Diabetes 70 - 140	mg/dL
METHOD : HEXOKINASE				
<b>CORONARY RISK PROFILE, SERUM</b>				
CHOLESTEROL		153	< 200 Desirable 200 - 239 Borderline High >/= 240 High	mg/dL
METHOD : CHOLESTEROL OXIDASE, ESTERASE, PEROXIDASE				
TRIGLYCERIDES		88	< 150 Normal 150 - 199 Borderline High 200 - 499 High >/= 500 Very High	mg/dL
METHOD : ENZYMATIC ASSAY				
HDL CHOLESTEROL		53	< 40 Low >/=60 High	mg/dL
METHOD : DIRECT MEASURE - PEG				
CHOLESTEROL LDL		82	< 100 Optimal 100 - 129 Near or above optimal 130 - 159 Borderline High 160 - 189 High >/= 190 Very High	mg/dL
METHOD : CHOLESTEROL OXIDASE, ESTERASE, PEROXIDASE				
NON HDL CHOLESTEROL		100	Desirable: Less than 130 Above Desirable: 130 - 159 Borderline High: 160 - 189 High: 190 - 219 Very high: > or = 220	mg/dL
METHOD : CALCULATED PARAMETER				
CHOL/HDL RATIO		<b>2.9</b>	<b>Low</b> 3.3-4.4 Low Risk 4.5-7.0 Average Risk 7.1-11.0 Moderate Risk > 11.0 High Risk	



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METHOD : CALCULATED PARAMETER				
LDL/HDL RATIO		1.5	0.5 - 3.0 Desirable/Low Risk 3.1 - 6.0 Borderline/Moderate Risk >6.0 High Risk	
METHOD : CALCULATED PARAMETER				
VERY LOW DENSITY LIPOPROTEIN		17.6	Desirable value : 10 - 35	mg/dL
METHOD : CALCULATED PARAMETER				
<b>LIVER FUNCTION PROFILE, SERUM</b>				
BILIRUBIN, TOTAL		1.09	UPTO 1.2	mg/dL
METHOD : DIAZONIUM ION, BLANKED ( ROCHE )				
BILIRUBIN, DIRECT		0.28	0.00 - 0.30	mg/dL
METHOD : DIAZOTIZATION				
BILIRUBIN, INDIRECT		<b>0.81</b>	<b>High</b> 0.00 - 0.60	mg/dL
METHOD : CALCULATED PARAMETER				
TOTAL PROTEIN		7.1	6.6 - 8.7	g/dL
METHOD : BIURET				
ALBUMIN		4.5	3.97 - 4.94	g/dL
METHOD : BROMOCRESOL GREEN				
GLOBULIN		2.6	2.0 - 4.0 Neonates - Pre Mature: 0.29 - 1.04	g/dL
METHOD : CALCULATED PARAMETER				
ALBUMIN/GLOBULIN RATIO		1.7	1.0 - 2.0	RATIO
METHOD : CALCULATED PARAMETER				
ASPARTATE AMINOTRANSFERASE (AST/SGOT)		20	0 - 40	U/L
ALANINE AMINOTRANSFERASE (ALT/SGPT)		15	0 - 41	U/L
METHOD : UV WITHOUT PYRIDOXAL-5 PHOSPHATE				
ALKALINE PHOSPHATASE		113	40 - 129	U/L
METHOD : PNPP - AMP BUFFER				
GAMMA GLUTAMYL TRANSFERASE (GGT)		9	8 - 61	U/L
METHOD : GAMMA GLUTAMYL CARBOXY 4NITROANILIDE				
LACTATE DEHYDROGENASE		162	135 - 225	U/L
METHOD : LACTATE -PYRUVATE				
<b>SERUM BLOOD UREA NITROGEN</b>				
BLOOD UREA NITROGEN		7	6 - 20	mg/dL
METHOD : UREASE - UV				
<b>CREATININE, SERUM</b>				
CREATININE		0.85	0.70 - 1.20	mg/dL



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METHOD : ALKALINE PICRATE-KINETIC

**BUN/CREAT RATIO**

BUN/CREAT RATIO 8.24 5.00 - 15.00

METHOD : CALCULATED PARAMETER

**URIC ACID, SERUM**

URIC ACID 5.6 3.4 - 7.0 mg/dL

METHOD : URICASE, COLORIMETRIC

**TOTAL PROTEIN, SERUM**

TOTAL PROTEIN 7.1 6.6 - 8.7 g/dL

METHOD : BIURET

**ALBUMIN, SERUM**

ALBUMIN 4.5 3.97 - 4.94 g/dL

METHOD : BROMOCRESOL GREEN

**GLOBULIN**

GLOBULIN 2.6 2.0 - 4.0 g/dL

Neonates -  
Pre Mature:  
0.29 - 1.04

METHOD : CALCULATED PARAMETER

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

SODIUM 140 136 - 145 mmol/L

METHOD : ISE INDIRECT

POTASSIUM 3.88 3.5 - 5.1 mmol/L

METHOD : ISE INDIRECT

CHLORIDE 107 98 - 107 mmol/L

METHOD : ISE INDIRECT

**PHYSICAL EXAMINATION, URINE**

COLOR PALE YELLOW

APPEARANCE CLEAR

SPECIFIC GRAVITY 1.015 1.003 - 1.035

METHOD : REFLECTANCE SPECTROPHOTOMETRY (PKA CHANGE OF PRETREATED POLY ELECTROLYTES)

**CHEMICAL EXAMINATION, URINE**

PH 6.0 4.7 - 7.5

METHOD : REFLECTANCE SPECTROPHOTOMETRY- DOUBLE INDICATOR METHOD

PROTEIN NOT DETECTED NOT DETECTED

METHOD : REFLECTANCE SPECTROPHOTOMETRY (PROTEIN-ERROR-OF-INDICATORS PRINCIPLE)

GLUCOSE NOT DETECTED NOT DETECTED

METHOD : REFLECTANCE SPECTROPHOTOMETRY (GLUCOSE OXIDASE/PEROXIDASE METHOD)



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KETONES		NOT DETECTED	NOT DETECTED	
METHOD : REFLECTANCE SPECTROPHOTOMETRY (SODIUM NITROPRUSSIDE REACTION)				
BLOOD		NOT DETECTED	NOT DETECTED	
METHOD : REFLECTANCE SPECTROPHOTOMETRY (PEROXIDASE METHOD)				
BILIRUBIN		NOT DETECTED	NOT DETECTED	
METHOD : REFLECTANCE SPECTROPHOTOMETRY (DIAZO REACTION)				
UROBILINOGEN		NORMAL	NORMAL	
METHOD : REFLECTANCE SPECTROPHOTOMETRY - EHRlich REACTION				
NITRITE		NOT DETECTED	NOT DETECTED	
METHOD : REFLECTANCE SPECTROPHOTOMETRY, CONVERSION OF NITRATE TO NITRITE				
LEUKOCYTE ESTERASE		NOT DETECTED	NOT DETECTED	
<b>MICROSCOPIC EXAMINATION, URINE</b>				
PUS CELL (WBC'S)		1-2	0-5	/HPF
METHOD : MICROSCOPIC EXAMINATION				
EPITHELIAL CELLS		1-2	0-5	/HPF
METHOD : MICROSCOPIC EXAMINATION				
ERYTHROCYTES (RBC'S)		NOT DETECTED	NOT DETECTED	/HPF
METHOD : MICROSCOPIC EXAMINATION				
CASTS		NOT DETECTED		
CRYSTALS		NOT DETECTED		
METHOD : MICROSCOPIC EXAMINATION				
BACTERIA		NOT DETECTED	NOT DETECTED	
METHOD : MICROSCOPIC EXAMINATION				
YEAST		NOT DETECTED	NOT DETECTED	
<b>THYROID PANEL, SERUM</b>				
T3		100.7	80.00 - 200.00	ng/dL
METHOD : COMPETITIVE (ECLIA)				
T4		6.44	5.10 - 14.10	µg/dL
METHOD : COMPETITIVE (ECLIA)				
TSH 3RD GENERATION		3.730	0.270 - 4.200	µIU/mL
METHOD : SANDWICH (ECLIA)				
<b>ABO GROUP &amp; RH TYPE, EDTA WHOLE BLOOD</b>				
ABO GROUP		TYPE O		
METHOD : TUBE AGGLUTINATION				
RH TYPE		POSITIVE		
METHOD : TUBE AGGLUTINATION				



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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 - NLR-

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.

## ERYTHRO SEDIMENTATION RATE, BLOOD-

Erythrocyte sedimentation rate (ESR) is a non-specific phenomena and is clinically useful in the diagnosis and monitoring of disorders associated with an increased production of acute phase reactants. The ESR is increased in pregnancy from about the 3rd month and returns to normal by the 4th week post partum. ESR is influenced by age, sex, menstrual cycle and drugs (eg. corticosteroids, contraceptives). It is especially low (0 -1mm) in polycythaemia, hypofibrinogenemia or congestive cardiac failure and when there are abnormalities of the red cells such as poikilocytosis, spherocytosis or sickle cells.

## Reference :

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

## GLYCOSYLATED HEMOGLOBIN, EDTA WHOLE BLOOD-

Glycosylated hemoglobin (GHb) has been firmly established as an index of long-term blood glucose concentrations and as a measure of the risk for the development of complications in patients with diabetes mellitus. Formation of GHb is essentially irreversible, and the concentration in the blood depends on both the life span of the red blood cell (average 120 days) and the blood glucose concentration. Because the rate of formation of GHb is directly proportional to the concentration of glucose in the blood, the GHb concentration represents the integrated values for glucose over the preceding 6-8 weeks.

Any condition that alters the life span of the red blood cells has the potential to alter the GHb level. Samples from patients with hemolytic anemias will exhibit decreased glycated hemoglobin values due to the shortened life span of the red cells. This effect will depend upon the severity of the anemia. Samples from patients with polycythemia or post-splenectomy may exhibit increased glycated hemoglobin values due to a somewhat longer life span of the red cells.

Glycosylated hemoglobins results from patients with HbSS, HbCC, and HbSC and HbD must be interpreted with caution, given the pathological processes, including anemia, increased red cell turnover, transfusion requirements, that adversely impact HbA1c as a marker of long-term glycemic control. In these conditions, alternative forms of testing such as glycated serum protein (fructosamine) should be considered.

"Targets should be individualized; More or less stringent glycemic goals may be appropriate for individual patients. Goals should be individualized based on duration of diabetes, age/life expectancy, comorbid conditions, known CVD or advanced microvascular complications, hypoglycemia unawareness, and individual patient considerations."

## References

1. Tietz Textbook of Clinical Chemistry and Molecular Diagnostics, edited by Carl A Burtis, Edward R. Ashwood, David E Bruns, 4th Edition, Elsevier publication, 2006, 879-884.
  2. Forsham PH. Diabetes Mellitus: A rational plan for management. Postgrad Med 1982, 71, 139-154.
  3. Mayer TK, Freedman ZR: Protein glycosylation in Diabetes Mellitus: A review of laboratory measurements and their clinical utility. Clin Chim Acta 1983, 127, 147-184.
- GLUCOSE, FASTING, PLASMA-  
ADA 2021 guidelines for adults, after 8 hrs fasting is as follows:  
Pre-diabetics: 100 - 125 mg/dL  
Diabetic: > or = 126 mg/dL  
GLUCOSE, POST-PRANDIAL, PLASMA-ADA Guidelines for 2hr post prandial glucose levels is only after ingestion of 75grams of glucose in 300 ml water, over a period of 5 minutes.

## 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 result 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



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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 globulin. Higher-than-normal levels may be due to: Chronic inflammation or infection, including HIV and hepatitis B or C, Multiple myeloma, Waldenstrom's disease. Lower-than-normal levels may be due to: Agammaglobulinemia, Bleeding (hemorrhage), Burns, Glomerulonephritis, Liver disease, Malabsorption, Malnutrition, Nephrotic syndrome, Protein-losing enteropathy etc. Human serum albumin is the most abundant protein in human blood plasma. It is produced in the liver. Albumin constitutes about half of the blood serum protein. Low blood albumin levels (hypoalbuminemia) can be caused by: Liver disease like cirrhosis of the liver, nephrotic syndrome, protein-losing enteropathy, Burns, hemodilution, increased vascular permeability or decreased lymphatic clearance, malnutrition and wasting etc

## SERUM BLOOD UREA NITROGEN-

Causes of Increased levels

Pre renal

- High protein diet, Increased protein catabolism, GI haemorrhage, Cortisol, Dehydration, CHF Renal

- Renal Failure

Post Renal

- Malignancy, Nephrolithiasis, Prostatism

Causes of decreased levels

- Liver disease

- SIADH.

CREATININE, SERUM-

Higher than normal level may be due to:

- Blockage in the urinary tract

- Kidney problems, such as kidney damage or failure, infection, or reduced blood flow

- Loss of body fluid (dehydration)

- Muscle problems, such as breakdown of muscle fibers

- Problems during pregnancy, such as seizures (eclampsia), or high blood pressure caused by pregnancy (preeclampsia)

Lower than normal level may be due to:

- Myasthenia Gravis

- Muscular dystrophy

URIC ACID, SERUM-

Causes of Increased levels

Dietary

- High Protein Intake.

- Prolonged Fasting,

- Rapid weight loss.

Gout

Lesch nyhan syndrome.

Type 2 DM.

Metabolic syndrome.

Causes of decreased levels

- Low Zinc Intake

- OCP's

- Multiple Sclerosis

Nutritional tips to manage increased Uric acid levels

- Drink plenty of fluids

- Limit animal proteins

- High Fibre foods

- Vit C Intake

- Antioxidant rich foods

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's disease

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

ALBUMIN, SERUM-



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CIN - U74899PB1995PLC045956

PATIENT NAME : NEPAL SINGH

PATIENT ID : NEPAM25086680

ACCESSION NO : 0080VI011002 AGE : 56 Years SEX : Male

ABHA NO :

DRAWN : RECEIVED : 24/09/2022 09:09

REPORTED : 25/09/2022 09:40

REFERRING DOCTOR : SELF

CLIENT PATIENT ID :

Test Report Status	Final	Results	Biological Reference Interval	Units
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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.

## ELECTROLYTES (NA/K/CL), SERUM-

Sodium levels are Increased in dehydration, cushing's syndrome, aldosteronism & decreased in Addison's disease, hypopituitarism, liver disease. Hypokalemia (low K) is common in vomiting, diarrhea, alcoholism, folic acid deficiency and primary aldosteronism. Hyperkalemia may be seen in end-stage renal failure, hemolysis, trauma, Addison's disease, metabolic acidosis, acute starvation, dehydration, and with rapid K infusion. Chloride is increased in dehydration, renal tubular acidosis (hyperchloremia metabolic acidosis), acute renal failure, metabolic acidosis associated with prolonged diarrhea and loss of sodium bicarbonate, diabetes insipidus, adrenocortical hyperfunction, salicylate intoxication and with excessive infusion of isotonic saline or extremely high dietary intake of salt. Chloride is decreased in overhydration, chronic respiratory acidosis, salt-losing nephritis, metabolic alkalosis, congestive heart failure, Addisonian crisis, certain types of metabolic acidosis, persistent gastric secretion and prolonged vomiting.

## MICROSCOPIC EXAMINATION, URINE-

Routine urine analysis assists in screening and diagnosis of various metabolic, urological, kidney and liver disorders

Protein: Elevated proteins can be an early sign of kidney disease. Urinary protein excretion can also be temporarily elevated by strenuous exercise, orthostatic proteinuria, dehydration, urinary tract infections and acute illness with fever

Glucose: Uncontrolled diabetes mellitus can lead to presence of glucose in urine. Other causes include pregnancy, hormonal disturbances, liver disease and certain medications.

Ketones: Uncontrolled diabetes mellitus can lead to presence of ketones in urine. Ketones can also be seen in starvation, frequent vomiting, pregnancy and strenuous exercise.

Blood: Occult blood can occur in urine as intact erythrocytes or haemoglobin, which can occur in various urological, nephrological and bleeding disorders.

Leukocytes: An increase in leukocytes is an indication of inflammation in urinary tract or kidneys. Most common cause is bacterial urinary tract infection.

Nitrite: Many bacteria give positive results when their number is high. Nitrite concentration during infection increases with length of time the urine specimen is retained in bladder prior to collection.

pH: The kidneys play an important role in maintaining acid base balance of the body. Conditions of the body producing acidosis/ alkalosis or ingestion of certain type of food can affect the pH of urine.

Specific gravity: Specific gravity gives an indication of how concentrated the urine is. Increased specific gravity is seen in conditions like dehydration, glycosuria and proteinuria while decreased specific gravity is seen in excessive fluid intake, renal failure and diabetes insipidus.

Bilirubin: In certain liver diseases such as biliary obstruction or hepatitis, bilirubin gets excreted in urine.

Urobilinogen: Positive results are seen in liver diseases like hepatitis and cirrhosis and in cases of hemolytic anemia

## THYROID PANEL, SERUM-

Triiodothyronine T3, is a thyroid hormone. It affects almost every physiological process in the body, including growth, development, metabolism, body temperature, and heart rate. Production of T3 and its prohormone thyroxine (T4) is activated by thyroid-stimulating hormone (TSH), which is released from the pituitary gland. Elevated concentrations of T3, and T4 in the blood inhibit the production of TSH.

Thyroxine T4, Thyroxine's principal function is to stimulate the metabolism of all cells and tissues in the body. Excessive secretion of thyroxine in the body is hyperthyroidism, and deficient secretion is called hypothyroidism. Most of the thyroid hormone in blood is bound to transport proteins. Only a very small fraction of the circulating hormone is free and biologically active.

In primary hypothyroidism, TSH levels are significantly elevated, while in secondary and tertiary hypothyroidism, TSH levels are low.

Below mentioned are the guidelines for Pregnancy related reference ranges for Total T4, TSH & Total T3

Levels in	TOTAL T4 (µg/dL)	TSH3G (µIU/mL)	TOTAL T3 (ng/dL)
Pregnancy	6.6 - 12.4	0.1 - 2.5	81 - 190
1st Trimester	6.6 - 15.5	0.2 - 3.0	100 - 260
2nd Trimester	6.6 - 15.5	0.3 - 3.0	100 - 260
3rd Trimester			

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

	T3 (ng/dL)	T4 (µg/dL)
New Born:	75 - 260	1-3 day: 8.2 - 19.9
		1 Week: 6.0 - 15.9

NOTE: TSH concentrations in apparently normal euthyroid subjects are known to be highly skewed, with a strong tailed distribution towards higher TSH values. This is well documented in the pediatric population including the infant age group.

Kindly note: Method specific reference ranges are appearing on the report under biological reference range.

## Reference:

1. Burtis C.A., Ashwood E. R. Bruns D.E. Teitz textbook of Clinical Chemistry and Molecular Diagnostics, 4th Edition.
2. Gowenlock A.H. Varley's Practical Clinical Biochemistry, 6th Edition.
3. Behrman R.E. Kilegman R.M., Jenson H. B. Nelson Text Book of Pediatrics, 17th Edition

## ABO GROUP &amp; 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.

**\*\*End Of Report\*\***

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**DIAGNOSTIC REPORT**

Patient Ref. No. 80000001326471



CLIENT CODE : C000138383

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**DR.CHANDNI GARG**  
CONSULTANT PATHOLOGIST

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