

8800465156

ACROFEMI HEALTHCARE LTD (MEDIWHEEL)
F-703, LADO SARAI, MEHRAULI
SOUTH WEST DELHI
NEW DELHI 110030
DELHI INDIA

SRL Ltd PLOT NO.160,POCKET D-11 SECTOR 8, ROHINI

NEW DELHI, 110085 NEW DELHI, INDIA Tel: 9111591115, Fax:

CIN - U74899PB1995PLC045956 Email : customercare.pitampura@srl.in

PATIENT NAME: ANJU PATIENT ID: ANJUF28108762

ACCESSION NO: 0062VJ000223 AGE: 34 Years SEX: Female ABHA NO:

DRAWN: RECEIVED: 08/10/2022 09:51:22 REPORTED: 10/10/2022 08:00:42

REFERRING DOCTOR: SELF CLIENT PATIENT ID:

Test Report Status	<u>Final</u>	Results		Biological Reference Interva	l Units
MEDI WIIFEL FILL DA	DDV HEALTH CHECKID D	TI OW 4055MALS			
BLOOD COUNTS,EDTA	ODY HEALTH CHECKUP BI N WHOLE BLOOD	ELOW 4UFEMALE			
HEMOGLOBIN	WHOLE BLOOD	12.0		12.0 - 15.0	g/dL
RED BLOOD CELL COUN	NT	3.94		3.8 - 4.8	g/αL mil/μL
WHITE BLOOD CELL CO		6.91		4.0 - 10.0	thou/µL
PLATELET COUNT	,0111	150		150 - 410	thou/μL
RBC AND PLATELET I	NDICES	150		130 410	τιου/ μΕ
HEMATOCRIT	NDICES	36.2		36 - 46	%
MEAN CORPUSCULAR V	/OI	91.9		83 - 101	fL
MEAN CORPUSCULAR H		30.5		27.0 - 32.0	pg
MEAN CORPUSCULAR H		33.2		31.5 - 34.5	g/dL
CONCENTRATION	ici io deobiiv	33.2		31.3 31.3	g/ uL
MENTZER INDEX		23.3			
RED CELL DISTRIBUTION	ON WIDTH	12.8		11.6 - 14.0	%
MEAN PLATELET VOLUM	1E	12.9	High	6.8 - 10.9	fL
WBC DIFFERENTIAL	COUNT - NLR				
SEGMENTED NEUTROPH	HILS	70		40 - 80	%
ABSOLUTE NEUTROPHI	L COUNT	4.84		2.0 - 7.0	thou/µL
LYMPHOCYTES		22		20 - 40	%
ABSOLUTE LYMPHOCYT	E COUNT	1.52		1 - 3	thou/µL
NEUTROPHIL LYMPHOC	YTE RATIO (NLR)	3.2			
EOSINOPHILS		03		1 - 6	%
ABSOLUTE EOSINOPHII	L COUNT	0.21		0.02 - 0.50	thou/µL
MONOCYTES		05		2 - 10	%
ABSOLUTE MONOCYTE	COUNT	0.35		0.20 - 1.00	thou/µL
BASOPHILS		00		0 - 2	%
ABSOLUTE BASOPHIL C	COUNT	0	Low	0.02 - 0.10	thou/µL
DIFFERENTIAL COUNT	PERFORMED ON:	EDTA SMEAR			
ERYTHRO SEDIMENT	ATION RATE, BLOOD				
SEDIMENTATION RATE METHOD: WESTERGREN ME	` '	50	High	0 - 20	mm at 1 hr

GLUCOSE, FASTING, PLASMA





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GLUCOSE, FASTING, PI	LASMA IETRY, O-CRESOLPHTHALEIN CO	85 DMPLEXONE		74 - 99	mg/dL
GLYCOSYLATED HEM	OGLOBIN, EDTA WHO	LE BLOOD			
GLYCOSYLATED HEMOO	GLOBIN (HBA1C)	5.1		Non-diabetic: < 5.7 Pre-diabetics: 5.7 - 6.4 Diabetics: > or = 6.5 ADA Target: 7.0 Action suggested: > 8.0	%
MEAN PLASMA GLUCOS	SE	99.7		< 116.0	mg/dL
GLUCOSE, POST-PRA	NDIAL, PLASMA				
GLUCOSE, POST-PRANI	DIAL, PLASMA	110		70 - 139	mg/dL
CORONARY RISK PRO	OFILE, SERUM				
CHOLESTEROL		293	High	< 200 Desirable 200 - 239 Borderline High >/= 240 High	mg/dL
METHOD: CHOD-POD				, 3	
TRIGLYCERIDES		260	High	< 150 Normal 150 - 199 Borderline High 200 - 499 High >/=500 Very High	mg/dL
METHOD : LIPASE / GLUCOS	SE DEHYDROGENASE	F7		4.40 1	/ -!!
HDL CHOLESTEROL		57		< 40 Low >/=60 High	mg/dL
CHOLESTEROL LDL		184	High	< 100 Optimal 100 - 129 Near optimal/ above optimal 130 - 159 Borderline High 160 - 189 High >/= 190 Very High	mg/dL
NON HDL CHOLESTERO	DL	236	High	Desirable: Less than 130 Above Desirable: 130 - 159 Borderline High: 160 - 189 High: 190 - 219 Very high: > or = 220	mg/dL







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CHOL/HDL RATIO	5.1	High	3.3 - 4.4 Low Risk	
			4.5 - 7.0	
			Average Risk 7.1 - 11.0	
			Moderate Risk	
			> 11.0 High Risk	
LDL/HDL RATIO	3.2	High	0.5 - 3.0 Desirable/Low Risk	
		_	3.1 - 6.0 Borderline/Moderate Risk >6.0 High Risk	
VERY LOW DENSITY LIPOPROTEIN	52.0	High	= 30.0</td <td>mg/dL</td>	mg/dL
LIVER FUNCTION PROFILE, SERUM				
BILIRUBIN, TOTAL	0.33		0.2 - 1.0	mg/dL
METHOD: SULPH ACID DPL/CAFF-BENZ				
BILIRUBIN, DIRECT	0.07		0.0 - 0.2	mg/dL
METHOD : SULPH ACID DPL/CAFF-BENZ				
BILIRUBIN, INDIRECT	0.26		0.1 - 1.0	mg/dL
METHOD: SPECTROPHOTOMETRY, MODIFIED DIAZO METHOD (3	ŕ			
TOTAL PROTEIN	6.9		6.4 - 8.2	g/dL
METHOD : SPECTROPHOTOMETRIC	2.5		2.4. 5.0	
ALBUMIN	3.5		3.4 - 5.0	g/dL
METHOD: SPECTROPHOTOMETRIC GLOBULIN	3.4		2.0 - 4.1	a /dl
METHOD : CALCULATED PARAMETER	3.4		2.0 - 4.1	g/dL
ALBUMIN/GLOBULIN RATIO	1.0		1.0 - 2.1	RATIO
METHOD : CALCULATED PARAMETER	1.0		1.0 2.1	104110
ASPARTATE AMINOTRANSFERASE (AST/SGOT)	27		15 - 37	U/L
METHOD : SPECTROPHOTOMETRIC-IFCC WITH UV WITH PYRIDO	XAL-5-PHOSPHATE			-,
ALANINE AMINOTRANSFERASE (ALT/SGPT)	38	High	< 34.0	U/L
METHOD: SPECTROPHOTOMETRIC-IFCC WITH UV WITH PYRIDO	XAL-5-PHOSPHATE			
ALKALINE PHOSPHATASE	129	High	30 - 120	U/L
METHOD: SPECTROPHOTOMETRIC				
GAMMA GLUTAMYL TRANSFERASE (GGT)	26		5 - 55	U/L
METHOD: SPECTROPHOTOMETRY, O-CRESOLPHTHALEIN COMPL	EXONE			
LACTATE DEHYDROGENASE	152		100 - 190	U/L
METHOD: SPECTROPHOTOMETRIC				







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SERUM BLOOD UREA	NITROGEN				
BLOOD UREA NITROGE	ΞN	8		6 - 20	mg/dL
METHOD : UREASE KINETIC					
CREATININE, SERUM	1				
CREATININE		0.63		0.60 - 1.10	mg/dL
METHOD : SPECTROPHOTON	METRY, O-CRESOLPHTHA	LEIN COMPLEXONE			
BUN/CREAT RATIO					
BUN/CREAT RATIO		12.70		5.00 - 15.00	
URIC ACID, SERUM					
URIC ACID		2.9		2.6 - 6.0	mg/dL
METHOD : URICASE/CATALA					
TOTAL PROTEIN, SEI	RUM				
TOTAL PROTEIN		6.9		6.4 - 8.2	g/dL
METHOD : BIURET					
ALBUMIN, SERUM					
ALBUMIN		3.5		3.4 - 5.0	g/dL
METHOD : SPECTROPHOTON	METRY, O-CRESOLPHTHA	LEIN COMPLEXONE			
GLOBULIN					
GLOBULIN		3.4		2.0 - 4.1	g/dL
METHOD : SPECTROPHOTON		LEIN COMPLEXONE			
ELECTROLYTES (NA/	(K/CL), SERUM				
SODIUM		129	Low	136 - 145	mmol/L
METHOD : ISE INDIRECT		2.06		2.50 5.40	1.0
POTASSIUM		3.86	_	3.50 - 5.10	mmol/L
CHLORIDE		96	Low	98 - 107	mmol/L
METHOD : ISE INDIRECT	TION UPINE				
PHYSICAL EXAMINA	IION, URINE				
COLOR		PALE YELLOW			
APPEARANCE		SLIGHTLY HAZY			
SPECIFIC GRAVITY		1.010		1.003 - 1.035	
CHEMICAL EXAMINA	TION, URINE				
PH		7.0		4.7 - 7.5	
PROTEIN		NOT DETECTED		NOT DETECTED	



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GLUCOSE	NOT DETECTED	NOT DETECTED		
KETONES	NOT DETECTED	NOT DETECTED		
BLOOD	NOT DETECTED	NOT DETECTED		
BILIRUBIN	NOT DETECTED	NOT DETECTED		
UROBILINOGEN	NORMAL	NORMAL		
NITRITE	NOT DETECTED	NOT DETECTED		
LEUKOCYTE ESTERASE	NOT DETECTED	NOT DETECTED		
MICROSCOPIC EXAMINATION, URINE				
PUS CELL (WBC'S)	10-15	0-5	/HPF	
EPITHELIAL CELLS	8-10	0-5	/HPF	
ERYTHROCYTES (RBC'S)	NOT DETECTED	NOT DETECTED	/HPF	
CASTS	NOT DETECTED			
CRYSTALS	NOT DETECTED			
BACTERIA	NOT DETECTED	NOT DETECTED		
YEAST	NOT DETECTED	NOT DETECTED		
REMARKS	NOTE:- MICROSCOPIO CENTRIFUGED URINARY SEDIMENT.			
THYROID PANEL, SERUM				
T3	147.30	80.00 - 200.00	ng/dL	
T4	11.01	5.10 - 14.10	μg/dL	
TSH 3RD GENERATION	3.920	0.270 - 4.200	μIU/mL	
PAPANICOLAOU SMEAR				
TEST METHOD	SAMPLE NOT RECEIVE	ED		
STOOL: OVA & PARASITE				
COLOUR	SAMPLE NOT RECEIVE	ED		
ABO GROUP & RH TYPE, EDTA WHOLE BL	.00D			
ABO GROUP	TYPE A			
METHOD: TUBE AGGLUTINATION				
RH TYPE	POSITIVE			



METHOD: TUBE AGGLUTINATION

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Interpretation(s)

BLOOD COUNTS, EDTA WHOLE BLOODThe 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.

WBC DIFFERENTIAL COUNT - NLRThe optimal threshold of 3.3 for NLR showed a prognostic possibility of clinical symptoms to change from mild to severe in COVID positive patients. When age = 49.5 years

old and NLR = 3.3, 46.1% COVID-19 patients with mild disease might become severe. By contrast, when age < 49.5 years old and NLR < 3.3, COVID-19 patients tend to show mild disease.

(Reference to - The diagnostic and predictive role of NLR, d-NLR and PLR in COVID-19 patients; A.-P. Yang, et al.; International Immunopharmacology 84 (2020) 106504 This ratio element is a calculated parameter and out of NABL scope.

ERYTHROCYTE SEDIMENTATION RATE (ESR), WHOLE BLOOD-TEST DESCRIPTION :-

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

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

TEST INTERPRETATION

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

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

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

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

Decreased in: Polycythermia vera, Sickle cell anemia

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,

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

GLUCOSE FASTING, FLUORIDE PLASMA-TEST DESCRIPTION

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

Increased in

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

Pancreatic islet cell disease with increased insulin,insulinoma,adrenocortical insufficiency, hypopituitarism,diffuse liver disease, malignancy (adrenocortical, stomach,fibrosarcoma), infant of a diabetic mother, enzyme deficiency diseases(e.g., galactosemia),Drugs- insulin, ethanol, propranolol; sulfonylureas,tolbutamide, and other oral hypoglycemic agents.

Hypoglycemia is defined as a glucoseof < 50 mg/dL in men and< 40 mg/dL in women.
While random serum glucose levels correlate with home glucose monitoring results (weekly mean capillary glucose values), there is wide fluctuation within individuals. Thus, glycosylated hemoglobin(HbA1c) levels are favored to monitor glycemic control.

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

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

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

The ADA recommends measurement of HbA1c (typically 3-4 times per year for type 1 and poorly controlled type 2 diabetic patients, and 2 times per year for well-controlled type 2 diabetic patients) to determine whether a patients metabolic control has remained continuously within the target range.

- 1.eAG (Estimated average glucose) converts percentage HbA1c to md/dl, to compare blood glucose levels.
- eAG gives an evaluation of blood glucose levels for the last couple of months.
 eAG is calculated as eAG (mg/dl) = 28.7 * HbA1c 46.7



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HbA1c Estimation can get affected due to :

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

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

IV.Interference of hemoglobinopathies in HbA1c estimation is seen in

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

c.HbF > 25% on alternate paltform (Boronate affinity chromatography) is recommended for testing of HbA1c.Abnormal Hemoglobin electrophoresis (HPLC method) is 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-

LIVER FUNCTION PROFILE

Bilirubin is a yellowish pigment found in bile and is a breakdown product of normal heme catabolism. Bilirubin is excreted in bile and urine, and elevated levels may give yellow discoloration in jaundice. Elevated levels results from increased bilirubin production (eg, hemolysis and ineffective erythropoiesis), decreased bilirubin excretion (eg, obstruction and hepatitis), and abnormal bilirubin metabolism (eg, hereditary and neonatal jaundice). Conjugated (direct) bilirubin is elevated more than unconjugated (indirect) bilirubin in Viral hepatitis, Drug reactions, Alcoholic liver disease Conjugated (direct) bilirubin is also elevated more than unconjugated (indirect) bilirubin when there is some kind of blockage of the bile ducts like in Gallstones getting into the bile ducts, tumors &Scarring of the bile ducts. Increased unconjugated (indirect) bilirubin may be a result of Hemolytic or pernicious anemia, Transfusion reaction & a common metabolic condition termed Gilbert syndrome, due to low levels of the enzyme that attaches sugar molecules to bilirubin.

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

ALP is a protein found in almost all body tissues. Tissues with higher amounts of ALP include the liver, bile ducts and bone. Elevated ALP levels are seen in Biliary obstruction, Osteoblastic bone tumors, osteomalacia, hepatitis, Hyperparathyroidism, Leukemia, Lymphoma, Paget's disease, Rickets, Sarcoidosis etc. Lower-than-normal ALP levels seen in Hypophosphatasia, Malnutrition, Protein deficiency, Wilson's disease. GGT is an enzyme found in cell membranes of many tissues mainly in the liver, kidney and pancreas. It is also found in other tissues including intestine, spleen, heart, brain and seminal vesicles. The highest concentration is in the kidney, but the liver is considered the source of normal enzyme activity. Serum GGT has been widely used as an index of liver dysfunction. Elevated serum GGT activity can be found in diseases of the liver, biliary system and pancreas. Conditions that increase serum GGT are obstructive liver disease, high alcohol consumption and use of enzyme-inducing drugs etc. Serum total protein, also known as total protein, is a biochemical test for measuring the total amount of protein in serum. Protein in the plasma is made up of albumin and globulin. Higher-than-normal levels may be due to: Chronic inflammation or infection, including HIV and hepatitis B or C, Multiple myeloma, Waldenstrom's disease. Lower-than-normal levels may be due to: Agammaglobulinemia, Bleeding (hemorrhage), Burns, Glomerulonephritis, Liver disease, Malabsorption, Malnutrition, Nephrotic syndrome, Protein-losing enteropathy etc. Human serum albumin is the most abundant protein in human blood plasma. It is produced in the liver. Albumin constitutes about half of the blood serum protein. Low blood albumin levels (hypoalbuminemia) can be caused by: Liver disease like cirrhosis of the liver, nephrotic syndrome, protein-losing enteropathy, Burns, hemodilution, increased vascular

permeability or decreased lymphatic clearance, malnutrition and wasting etc BLOOD UREA NITROGEN (BUN), SERUM-Causes of Increased levels include Pre renal (High protein diet, Increased protein catabolism, GI haemorrhage, Cortisol, Dehydration, CHF Renal), Renal Failure, Post Renal (Malignancy, Nephrolithiasis, Prostatism)

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

- Blockage in the urinary tract
 Kidney problems, such as kidney damage or failure, infection, or reduced blood flow
- Loss of body fluid (dehydration)
- Muscle problems, such as breakdown of muscle fibers
- Problems during pregnancy, such as seizures (eclampsia)), or high blood pressure caused by pregnancy (preeclampsia)

Lower than normal level may be due to:

- Myasthenia Gravis
- Muscular dystrophy URIC ACID, SERUM-

Causes of Increased levels

Dietary

- High Protein Intake.
- Prolonged Fasting,
- Rapid weight loss.

Gout

Lesch nyhan syndrome.



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Type 2 DM. Metabolic syndrome.

Causes of decreased levels

- Low Zinc IntakeOCP's
- Multiple Sclerosis

Nutritional tips to manage increased Uric acid levels

- Drink plenty of fluids
- Limit animal proteins
- High Fibre foods
- Vit C IntakeAntioxidant 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

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.

ELECTROLYTES (NA/K/CL), SERUMSodium 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 hyperfuction, 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

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 ΤΟΤΔΙ Τ4 TSH3G TOTAL T3 (µg/dL) Pregnancy (µIU/mL) (ng/dL)







ACROFEMI HEALTHCARE LTD (MEDIWHEEL)

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

DELHI INDIA 8800465156

SRL Ltd

PLOT NO.160, POCKET D-11 SECTOR 8, ROHINI

NEW DELHI, 110085 NEW DELHI, INDIA Tel: 9111591115, Fax:

CIN - U74899PB1995PLC045956 Email: customercare.pitampura@srl.in

PATIENT NAME: ANJU PATIENT ID: ANJUF28108762

0062VJ000223 AGE: 34 Years SEX: Female ABHA NO: ACCESSION NO:

DRAWN: RECEIVED: 08/10/2022 09:51:22 REPORTED: 10/10/2022 08:00:42

REFERRING DOCTOR: SFIF CLIENT PATIENT ID:

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

First Trimester 6.6 - 12.4 0.1 - 2.5 81 - 190 0.2 - 3.0 0.3 - 3.0 100 - 260 100 - 260 2nd Trimester 6.6 - 15.5 6.6 - 15.5 3rd Trimester

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

T3 (μg/dL) 1-3 day: 8.2 - 19.9 (ng/dL) New Born: 75 - 260 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
 STOOL: OVA & PARASITE-

Acute infective diarrhoea and gastroenteritis (diarrhoea with vomiting) are major causes of ill health and premature death in developing countries. Loss of water and electrolytes from the body can lead to severe dehydration which if untreated, can be rapidly fatal in young children, especially that are malnourished, hypoglycaemic, and generally in poor health.

Laboratory diagnosis of parasitic infection is mainly based on microscopic examination and the gross examination of the stool specimen. Depending on the nature of the parasite, the microscopic observations include the identification of cysts, ova, trophozoites, larvae or portions of adult structure. The two classes of parasites that cause human infection are the Protozoa and Helminths. The protozoan infections include amoebiasis mainly caused by Entamoeba histolytica and giardiasis caused by Giardia lamblia. The common helminthic parasites are Trichuris trichiura, Ascaris lumbricoides, Strongyloides stercoralis, Taenia sp. etc ABO GROUP & RH TYPE, EDTA WHOLE BLOOD-

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

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

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

End Of Report

Please visit www.srlworld.com for related Test Information for this accession

Dr. Kamlesh I Prajapati **Consultant Pathologist**



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