





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 : GURVINDER SI	PATIENT ID : GURVM06059027	
ACCESSION NO : 0080VI011006	AGE : 32 Years SEX : Male	ABHA NO :
DRAWN :	RECEIVED : 24/09/2022 09:14	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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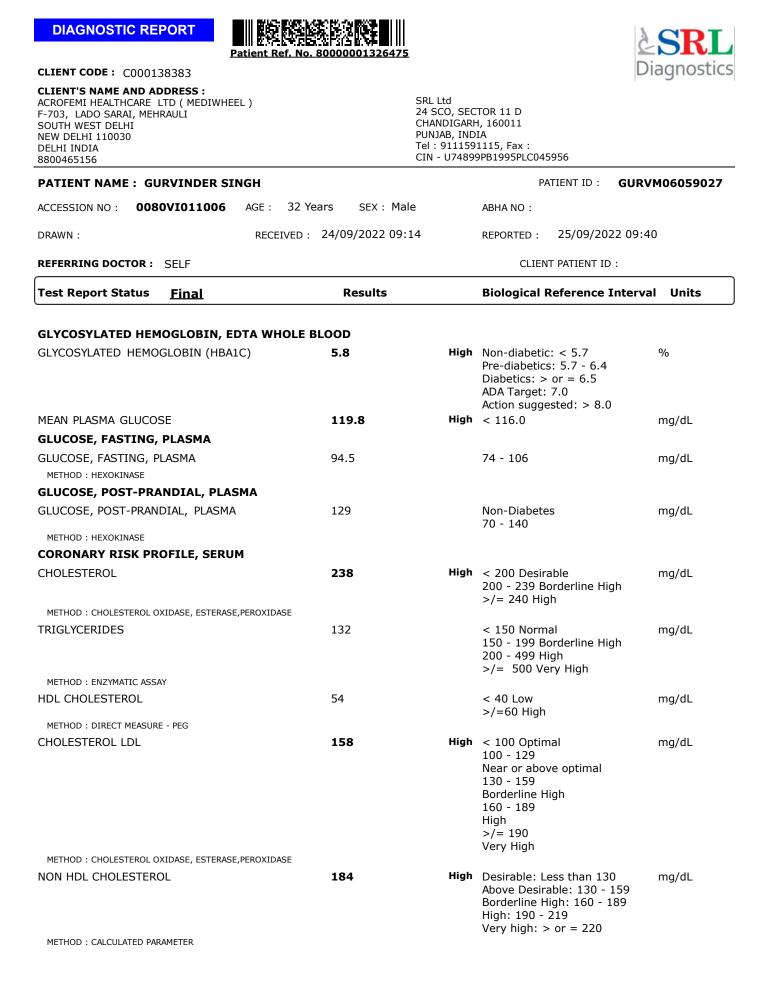
MEDI WHEEL FULL BODY HEALTH CHECK UP ABOVE 40 MALE

BLOOD COUNTS, EDTA WHOLE BLOOD				
HEMOGLOBIN	14.5		13.0 - 17.0	g/dL
RED BLOOD CELL COUNT	5.06 4.1		4.5 - 5.5	mil/µL
WHITE BLOOD CELL COUNT	6.70		4.0 - 10.0	thou/µL
PLATELET COUNT	291		150 - 410	thou/µL
RBC AND PLATELET INDICES				
HEMATOCRIT	43.3		40.0 - 50.0	%
METHOD : ELECTRICAL IMPEDANCE				
MEAN CORPUSCULAR VOL	85.7		83.0 - 101.0	fL
MEAN CORPUSCULAR HGB.	28.6		27.0 - 32.0	pg
MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION	33.4		31.5 - 34.5	g/dL
MENTZER INDEX	16.9			
RED CELL DISTRIBUTION WIDTH	12.7		11.6 - 14.0	%
MEAN PLATELET VOLUME	8.7		6.8 - 10.9	fL
WBC DIFFERENTIAL COUNT - NLR				
SEGMENTED NEUTROPHILS	56		40 - 80	%
ABSOLUTE NEUTROPHIL COUNT	3.75		2.0 - 7.0	thou/µL
LYMPHOCYTES	33		20 - 40	%
ABSOLUTE LYMPHOCYTE COUNT	2.21		1.0 - 3.0	thou/µL
NEUTROPHIL LYMPHOCYTE RATIO (NLR) METHOD : CALCULATED PARAMETER	1.8			
EOSINOPHILS	4		1 - 6	%
ABSOLUTE EOSINOPHIL COUNT	0.27		0.02 - 0.50	thou/µL
MONOCYTES	7		2 - 10	%
ABSOLUTE MONOCYTE COUNT METHOD : CALCULATED PARAMETER	0.47		0.2 - 1.0	thou/µL
BASOPHILS	0		0 - 1	%
ABSOLUTE BASOPHIL COUNT METHOD : CALCULATED PARAMETER	0.00	Low	0.02 - 0.10	thou/µL
DIFFERENTIAL COUNT PERFORMED ON:	AUTOMATED ANALY	ZER		
ERYTHRO SEDIMENTATION RATE, BLOOD				
SEDIMENTATION RATE (ESR)	24	High	0 - 14	mm at 1 hr

METHOD : MODIFIED WESTERGREN

















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Test Report Status <u>Final</u>	Results		Biological Reference In	iterval	Units
CHOL/HDL RATIO	4.4		3.3-4.4 Low Risk 4.5-7.0 Average Risk 7.1-11.0 Moderate Risk > 11.0 High Risk		
METHOD : CALCULATED PARAMETER	2.9		0.5 - 3.0 Desirable/Low R 3.1 - 6.0 Borderline/Mode >6.0 High Risk		sk
METHOD : CALCULATED PARAMETER VERY LOW DENSITY LIPOPROTEIN	26.4		Desirable value : 10 - 35		mg/dL
METHOD : CALCULATED PARAMETER					
LIVER FUNCTION PROFILE, SERUM					
BILIRUBIN, TOTAL METHOD : DIAZONIUM ION, BLANKED (ROCHE)	0.58		UPTO 1.2	l	mg/dL
BILIRUBIN, DIRECT METHOD : DIAZOTIZATION	0.17		0.00 - 0.30		mg/dL
BILIRUBIN, INDIRECT METHOD : CALCULATED PARAMETER	0.41		0.00 - 0.60	l	mg/dL
TOTAL PROTEIN METHOD : BIURET	8.3		6.6 - 8.7		g/dL
ALBUMIN METHOD : BROMOCRESOL GREEN	5.1	High	3.97 - 4.94		g/dL
GLOBULIN	3.2		2.0 - 4.0 Neonates - Pre Mature: 0.29 - 1.04		g/dL
METHOD : CALCULATED PARAMETER					
ALBUMIN/GLOBULIN RATIO METHOD : CALCULATED PARAMETER	1.6		1.0 - 2.0		RATIO
ASPARTATE AMINOTRANSFERASE (AST/SGOT)	34		0 - 40		U/L
ALANINE AMINOTRANSFERASE (ALT/SGPT) METHOD : UV WITHOUT PYRIDOXAL-5 PHOSPHATE	63	High	0 - 41		U/L
ALKALINE PHOSPHATASE METHOD : PNPP - AMP BUFFER	76		40 - 129		U/L
GAMMA GLUTAMYL TRANSFERASE (GGT) METHOD : GAMMA GLUTAMYLCARBOXY 4NITROANILIDE	47		8 - 61		U/L
LACTATE DEHYDROGENASE METHOD : LACTATE -PYRUVATE	175		135 - 225		U/L
SERUM BLOOD UREA NITROGEN					
BLOOD UREA NITROGEN	9		6 - 20		mg/dL









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Test Report Status	<u>Final</u>	Results		Biological Reference Inte	erval Units
METHOD : UREASE - UV					
CREATININE, SERUM	М				
CREATININE		0.90		0.70 - 1.20	mg/dL
METHOD : ALKALINE PICRA	TE-KINETIC				
BUN/CREAT RATIO					
BUN/CREAT RATIO		10.00		5.00 - 15.00	
METHOD : CALCULATED PA	RAMETER				
URIC ACID, SERUM					
URIC ACID		6.4		3.4 - 7.0	mg/dL
METHOD : URICASE, COLOR	RIMETRIC				
TOTAL PROTEIN, SE	RUM				
TOTAL PROTEIN		8.3		6.6 - 8.7	g/dL
METHOD : BIURET					
ALBUMIN, SERUM					
ALBUMIN		5.1	High	3.97 - 4.94	g/dL
METHOD : BROMOCRESOL	GREEN				
GLOBULIN					
GLOBULIN		3.2		2.0 - 4.0 Neonates - Pre Mature: 0.29 - 1.04	g/dL
METHOD : CALCULATED PA					
ELECTROLYTES (NA)	/K/CL), SERUM				
SODIUM		138		136 - 145	mmol/L
METHOD : ISE INDIRECT					
POTASSIUM		4.42		3.5 - 5.1	mmol/L
METHOD : ISE INDIRECT		102		00 107	
CHLORIDE METHOD : ISE INDIRECT		103		98 - 107	mmol/L
PHYSICAL EXAMINA					
COLOR	TION, ORINE	PALE YELLOW			
		CLEAR			
APPEARANCE				1 002 1 025	
SPECIFIC GRAVITY				1.003 - 1.035	
CHEMICAL EXAMINA		IGE OF PRETREATED POLY ELECTRO	LIIL3)		
PH		6.0		4.7 - 7.5	
	PECTROPHOTOMETRY- DOUBLE I			т./ ⁻ /.J	
PROTEIN	Lettor not one tru- DOUBLE I	NOT DETECTED		NOT DETECTED	

8800465156 **PATIENT NAME : GURVINDER SINGH**

DRAWN :

ACCESSION NO : 0080VI011006

DIAGNOSTIC REPORT

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UU 171



AGE : 32 Years SEX : Male

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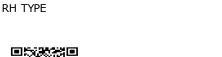
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Test Report Status <u>Final</u>	Results	Biological Reference Interval	Units
METHOD : REFLECTANCE SPECTROPHOTOMETRY (PROTEIN-ERROF		NOT DETECTED	
		NOT DETECTED	
METHOD : REFLECTANCE SPECTROPHOTOMETRY(GLUCOSE OXIDA			
KETONES		NOT DETECTED	
METHOD : REFLECTANCE SPECTROPHOTOMETRY (SODIUM NITRO			
	NOT DETECTED	NOT DETECTED	
METHOD : REFLECTANCE SPECTROPHOTOMETRY (PEROXIDASE ME			
	NOT DETECTED	NOT DETECTED	
METHOD : REFLECTANCE SPECTROPHOTOMETRY (DIAZO REACTIO	NORMAL	NORMAL	
UROBILINOGEN METHOD : REFLECTANCE SPECTROPHOTOMETRY - EHRLICH REAC		NORMAL	
NITRITE	NOT DETECTED	NOT DETECTED	
METHOD : REFLECTANCE SPECTROPHOTOMETRY, CONVERSION O		NOT DETECTED	
LEUKOCYTE ESTERASE	NOT DETECTED	NOT DETECTED	
	NOT DETECTED	NOT DETECTED	
	1.2	a F	(1105
PUS CELL (WBC'S)	1-2	0-5	/HPF
	0.1		
EPITHELIAL CELLS	0-1	0-5	/HPF
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		NOT DETECTED	
YEAST	NOT DETECTED	NOT DETECTED	
THYROID PANEL, SERUM			
Т3	130.0	80.00 - 200.00	ng/dL
METHOD : COMPETITIVE (ECLIA)			
T4	8.76	5.10 - 14.10	µg/dL
METHOD : COMPETITIVE (ECLIA)			
TSH 3RD GENERATION	2.690	0.270 - 4.200	µIU/mL
METHOD : SANDWICH (ECLIA)			
ABO GROUP & RH TYPE, EDTA WHOLE BLOOD			
ABO GROUP	TYPE A		
METHOD : TUBE AGGLUTINATION			

POSITIVE











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METHOD : TUBE AGGLUTINATION

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 be a severe. 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

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 early them and the mediative for the set of 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. 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" 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

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 75 grams 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 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 is viral hepatitis, Drug reactions, Alcoholic liver disease Conjugated (direct) bilirubin is also elevated more than unconjugated (indirect) bilirubin viral hepatitis). 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.









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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 Agamaglobulinemia, 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 proteinsHigh Fibre foods

Vit C Intake

Antioxidant rich foods

TOTAL PROTEIN, SERUM-

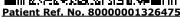
Serum total protein, also known as total protein, is a biochemical test for measuring the total amount of protein in serum.. Protein in the plasma is made up of albumin and globulin

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



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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.

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 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 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.

al T4, TSH & Total T3

Below mentioned	are the guidelines for	or Pregnancy related	reference ranges for	· Tota
Levels in	TOTAL T4	TSH3G	TOTAL T3	
Pregnancy	(µg/dL)	(µIU/mL)	(ng/dL)	
First Trimester	6.6 - 12.4	0.1 - 2.5	81 - 190	
and Trimoctor	66 155	0 2 2 0	100 260	

ringe rinnegter	0.0 12.1	0.1 2.3	01 190	
2nd Trimester	6.6 - 15.5	0.2 - 3.0	100 - 260	
3rd Trimester	6.6 - 15.5	0.3 - 3.0	100 - 260	
Below mentioned	are the guidelines f	or age related refere	nce ranges for T3 and T4	ŀ.,

13	14
(ng/dL)	(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.

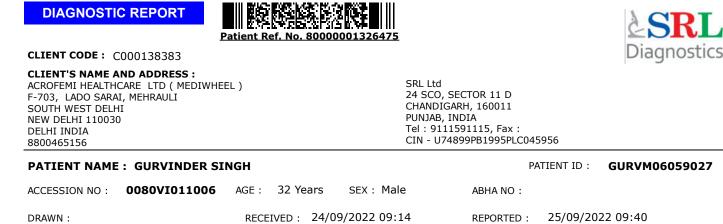
Gowenlock A.H. Varley's Practical Clinical Biochemistry, 6th Edition.
 Behrman R.E. Kilegman R.M., Jenson H. B. Nelson Text Book of Pediatrics, 17th Edition

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.







REFERRING DOCTOR : SELF

CLIENT PATIENT ID :

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

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

Chandni Garg

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Pravalit

Dr.Pranjali Vasisht LAB HEAD



