DIAGNOSTIC REPORT

agilus diagnostics

PATIENT NAME : PREETPAL SINGH S/O S.AMRIK SINGH **REF. DOCTOR :** SELF

PREETPAL SINGH S/O S.AMRIK SINGH
AMRITSAR
Amritsar 143001

ACCESSION NO : **0202WJ007460**
PATIENT ID : PREEM19027978A
CLIENT PATIENT ID:
ABHA NO :

AGE/SEX :44 Years Male
DRAWN :21/10/2023 12:36:27
RECEIVED :21/10/2023 12:39:49
REPORTED :21/10/2023 14:56:46

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

HAEMATOLOGY - CBC

COMPLETE CARE TOTAL WITH SMART REPORT

BLOOD COUNTS,EDTA WHOLE BLOOD

HEMOGLOBIN (HB) METHOD : CYANMETHEMOGLOBIN METHOD	14.5	13.0 - 17.0	g/dL
RED BLOOD CELL (RBC) COUNT METHOD : ELECTRICAL IMPEDANCE	5.01	4.5 - 5.5	mil/µL
WHITE BLOOD CELL (WBC) COUNT METHOD : ELECTRICAL IMPEDANCE	**14.10 High**	4.0 - 10.0	thou/µL
PLATELET COUNT METHOD : ELECTRONIC IMPEDANCE/CALCULATION	296	150 - 410	thou/µL

RBC AND PLATELET INDICES

HEMATOCRIT (PCV) METHOD : ELECTRICAL IMPEDANCE	43.9	40 - 50	%
MEAN CORPUSCULAR VOLUME (MCV) METHOD : CALCULATED PARAMETER	88.0	83 - 101	fL
MEAN CORPUSCULAR HEMOGLOBIN (MCH) METHOD : CALCULATED PARAMETER	28.9	27.0 - 32.0	pg
MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION (MCHC) METHOD : CALCULATED PARAMETER	32.9	31.5 - 34.5	g/dL
RED CELL DISTRIBUTION WIDTH (RDW) METHOD : CALCULATED PARAMETER	**14.3 High**	11.6 - 14.0	%
MENTZER INDEX	17.6		
MEAN PLATELET VOLUME (MPV) METHOD : CALCULATED PARAMETER	8.1	6.8 - 10.9	fL

WBC DIFFERENTIAL COUNT

NEUTROPHILS METHOD : ELECTRICAL IMPEDANCE	62	40 - 80	%
LYMPHOCYTES METHOD : ELECTRICAL IMPEDANCE	32	20 - 40	%
MONOCYTES METHOD : ELECTRICAL IMPEDANCE	4	2 - 10	%
EOSINOPHILS METHOD : ELECTRICAL IMPEDANCE	2	1 - 6	%

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Tel : 9111591115, Fax : CIN - U74899PB1995PLC045956

Patient Ref. No. 775000005180288

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BASOPHILS	0	0 - 2	%
METHOD : ELECTRICAL IMPEDANCE			
ABSOLUTE NEUTROPHIL COUNT	**8.74 High**	2.0 - 7.0	thou/µL
ABSOLUTE LYMPHOCYTE COUNT	**4.51 High**	1.0 - 3.0	thou/µL
ABSOLUTE MONOCYTE COUNT	0.56	0.2 - 1.0	thou/µL
ABSOLUTE EOSINOPHIL COUNT	0.28	0.02 - 0.50	thou/µL
ABSOLUTE BASOPHIL COUNT	**0.00 Low**	0.02 - 0.10	thou/µL
NEUTROPHIL LYMPHOCYTE RATIO (NLR)	1.5		
METHOD : CALCULATED			

PERIPHERAL SMEAR EXAM, EDTA WHOLE BLOOD

RBC	RBC ARE PREDOMINANTLY NORMOCYTIC NORMOCHROMIC. MILD ANISOPOIKILOCYTOSIS IS SEEN. NO NUCLEATED RBC SEEN.POLYCHROMASIA IS NOT RAISED.
METHOD : MICROSCOPIC EXAMINATION	
WBC	TOTAL LEUCOCYTE COUNT IS RAISED. DIFFERENTIAL COUNT SHOWS ABSOLUTE NEUTROPHILIA AND ABSOLUTE LYMPHOCYTOSIS
METHOD : MICROSCOPIC EXAMINATION	
PLATELETS	PLATELETS ARE ADEQUATE IN NUMBER
METHOD : MICROSCOPIC EXAMINATION	
IMPRESSION	NORMOCYTIC NORMOCHROMIC SMEAR WITH LEUCOCYTOSIS ABSOLUTE NEUTROPHILIA AND ABSOLUTE LYMPHOCYTOSIS. KINDLY CORRELATE CLINICALLY.

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-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.

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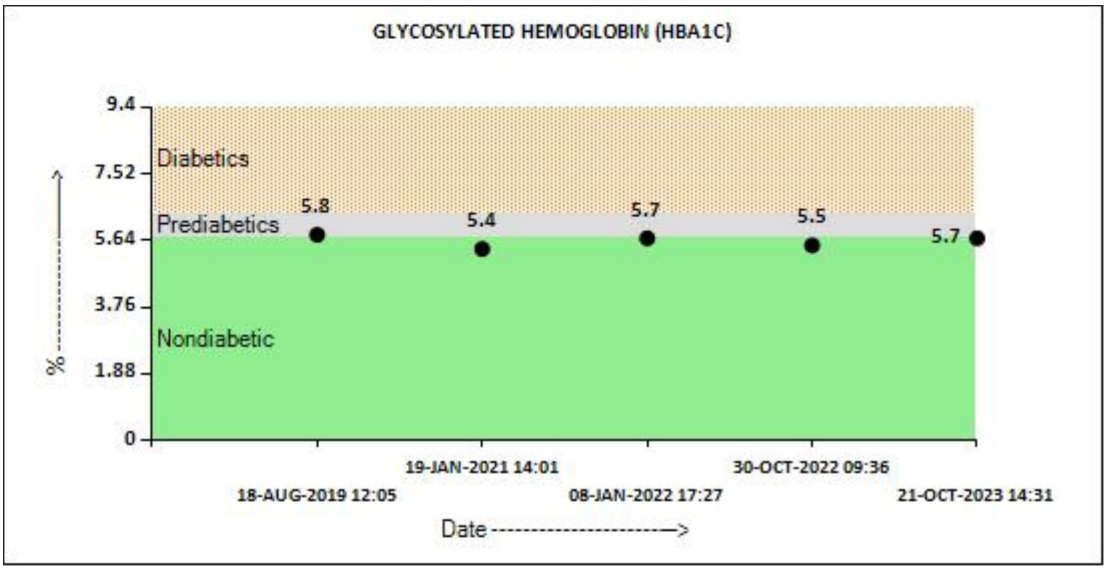
HAEMATOLOGY

COMPLETE CARE TOTAL WITH SMART REPORT

ERYTHROCYTE SEDIMENTATION RATE (ESR),WHOLE BLOOD			
E.S.R	03	0 - 14	mm at 1 hr
METHOD : MODIFIED WESTERGREN			
GLYCOSYLATED HEMOGLOBIN(HBA1C), EDTA WHOLE BLOOD			
HBA1C	5.7	Non-diabetic: < 5.7 Pre-diabetics: 5.7 - 6.4 Diabetics: > or = 6.5 Therapeutic goals: < 7.0 Action suggested : > 8.0 (ADA Guideline 2021)	%
METHOD : HIGH PERFORMANCE LIQUID CHROMATOGRAPHY (HPLC)			
ESTIMATED AVERAGE GLUCOSE(EAG)	**116.9 High**	< 116.0	mg/dL
METHOD : CALCULATED PARAMETER			



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

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

Increase in: Infections, Vasculities, Inflammatory arthritis, Renal disease, Anemia, Malignancies and plasma cell dyscrasias, Acute allergy Tissue injury, Pregnancy, Estrogen medication, Aging.
Finding a very accelerated ESR**(>100 mm/hour)** in patients with ill-defined symptoms directs the physician to search for a systemic disease (Paraproteinemias, Disseminated malignancies, connective tissue disease, severe infections such as bacterial endocarditis).
In pregnancy BRI in first trimester is 0-48 mm/hr(62 if anemic) and in second trimester (0-70 mm /hr(95 if anemic). ESR returns to normal 4th week post partum.
Decreased in: Polycythermia vera, Sickle cell anemia

LIMITATIONS

False elevated ESR : Increased fibrinogen, Drugs(Vitamin A, Dextran etc), Hypercholesterolemia
False Decreased : Poikilocytosis,(SickleCells,spherocytes),Microcytosis, Low fibrinogen, Very high WBC counts, Drugs(Quinine,

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.
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.
2. eAG gives an evaluation of blood glucose levels for the last couple of months.
3. eAG is calculated as eAG (mg/dl) = 28.7 * HbA1c - 46.7

HbA1c Estimation can get affected due to :
1. Shortened Erythrocyte survival : Any condition that shortens erythrocyte survival or decreases mean erythrocyte age (e.g. recovery from acute blood loss,hemolytic anemia) will falsely lower HbA1c test results.Fructosamine is recommended in these patients which indicates diabetes control over 15 days.
2.Vitamin C & E are reported to falsely lower test results.(possibly by inhibiting glycation of hemoglobin.
3. Iron deficiency anemia is reported to increase test results. Hypertriglyceridemia,uremia, hyperbilirubinemia, chronic alcoholism,chronic ingestion of salicylates & opiates addiction are reported to interfere with some assay methods,falsely increasing results.
4. Interference of hemoglobinopathies in HbA1c estimation is seen in

a) Homozygous hemoglobinopathy. Fructosamine is recommended for testing of HbA1c.
b) Heterozygous state detected (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

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BIOCHEMISTRY

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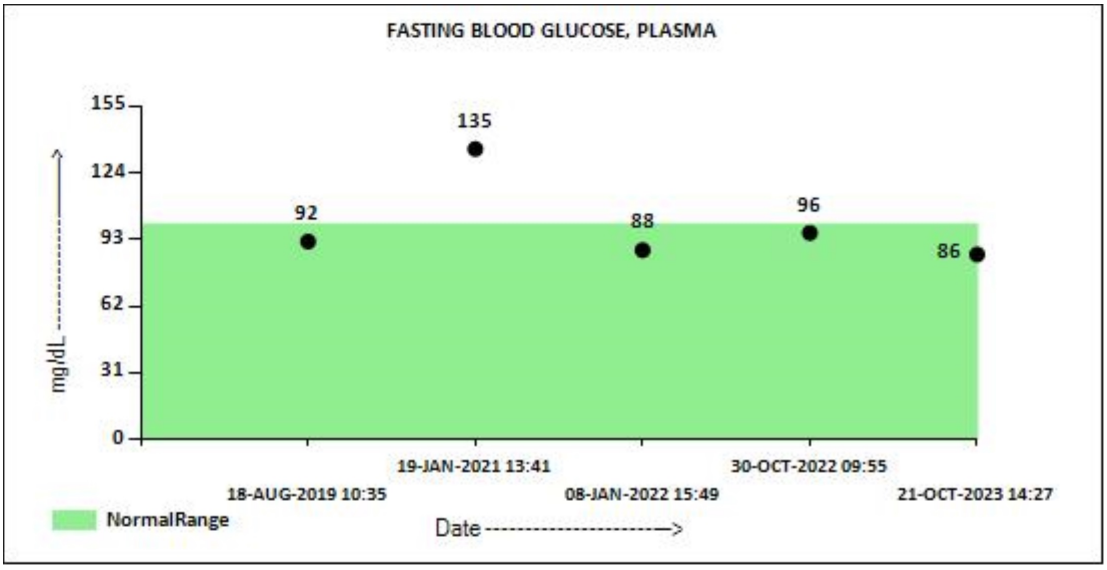
GLUCOSE FASTING,FLUORIDE PLASMA

FBS (FASTING BLOOD SUGAR)	86	Normal : < 100 Pre-diabetes: 100-125 Diabetes: >/=126	mg/dL

METHOD : HEXOKINASE



LIVER FUNCTION PROFILE, SERUM

BILIRUBIN, TOTAL	0.37	0.2 - 1.0	mg/dL
METHOD : DIAZOTIZED SULFANULIC ACD / CAFFEINE			
BILIRUBIN, DIRECT	0.08	0.0 - 0.2	mg/dL
METHOD : DIAZO WITH SULPHANILIC ACID			
BILIRUBIN, INDIRECT	0.29	0.1 - 1.0	mg/dL
METHOD : CALCULATED PARAMETER			
TOTAL PROTEIN	6.9	6.4 - 8.2	g/dL
METHOD : BIURET			
ALBUMIN	3.9	3.4 - 5.0	g/dL
METHOD : BCG DYE BINDING METHOD			

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GLOBULIN METHOD : CALCULATED PARAMETER	3.0	2.0 - 4.1	g/dL
ALBUMIN/GLOBULIN RATIO METHOD : CALCULATED PARAMETER	1.3	1.0 - 2.1	RATIO
ASPARTATE AMINOTRANSFERASE(AST/SGOT) METHOD : ASPARTIC ACID, PYRIDOXAL 5 PHOSPHATE / UV	25	15 - 37	U/L
ALANINE AMINOTRANSFERASE (ALT/SGPT) METHOD : ALANINE, PYRIDOXAL 5 PHOSPHATE / LDH	**46 High**	< 45.0	U/L
ALKALINE PHOSPHATASE METHOD : PNPP - AMP BUFFER	82	30 - 120	U/L
GAMMA GLUTAMYL TRANSFERASE (GGT) METHOD : GCNA	28	15 - 85	U/L
LACTATE DEHYDROGENASE METHOD : L-LACTATE, NAD / UV	161	85 - 227	U/L
LIPID PROFILE WITH CALCULATED LDL			
CHOLESTEROL, TOTAL METHOD : CHOLESTEROL ESTERASE (CE) / CHOLESTEROL OXIDASE (CO)	189	< 200 Desirable 200 - 239 Borderline High >/= 240 High	mg/dL
TRIGLYCERIDES METHOD : LIPOPROTEIN LIPASE (LPL), GLYCEROL KINASE (GK)	**228 High**	< 150 Normal 150 - 199 Borderline High 200 - 499 High >/=500 Very High	mg/dL
HDL CHOLESTEROL METHOD : PEG MODIFIED CHOLESTEROL ESTERASE AND CHOLESTEROL OXIDASE	**36 Low**	< 40 Low >/=60 High	mg/dL
CHOLESTEROL LDL METHOD : DIRECT HOMOGENOUS	**107 High**	< 100 Optimal 100 - 129 Near optimal/ above optimal 130 - 159 Borderline High 160 - 189 High >/= 190 Very High	mg/dL
NON HDL CHOLESTEROL	**153 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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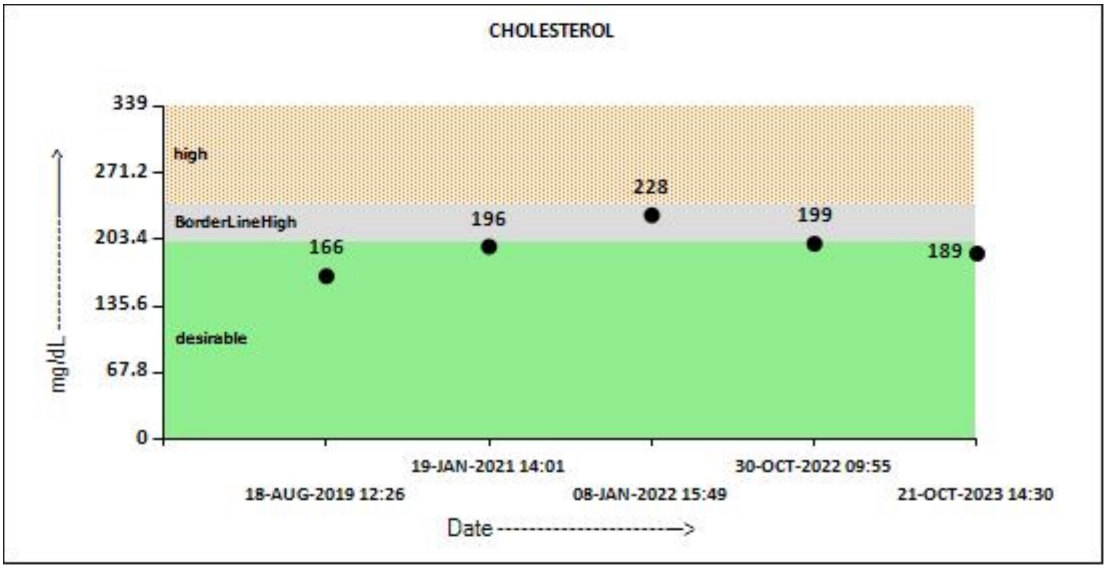
VERY LOW DENSITY LIPOPROTEIN METHOD : CALCULATED PARAMETER	**45.6 High**	</= 30.0	mg/dL
CHOL/HDL RATIO METHOD : CALCULATED PARAMETER	**5.3 High**	3.3 - 4.4 Low Risk 4.5 - 7.0 Average Risk 7.1 - 11.0 Moderate Risk > 11.0 High Risk	
LDL/HDL RATIO METHOD : CALCULATED PARAMETER	3.0	0.5 - 3.0 Desirable/Low Risk 3.1 - 6.0 Borderline/Moderate Risk >6.0 High Risk	



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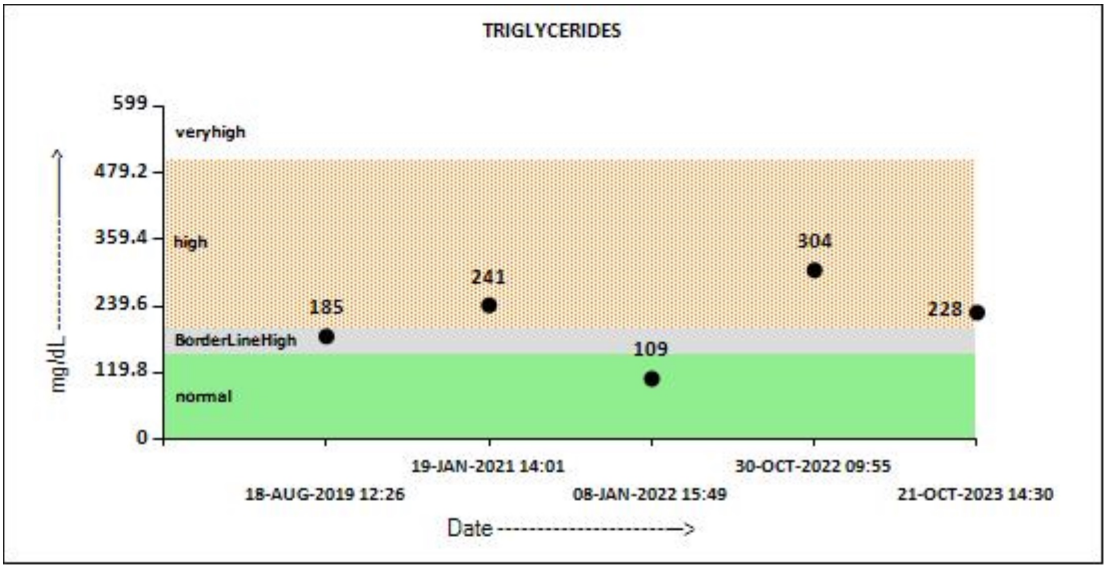

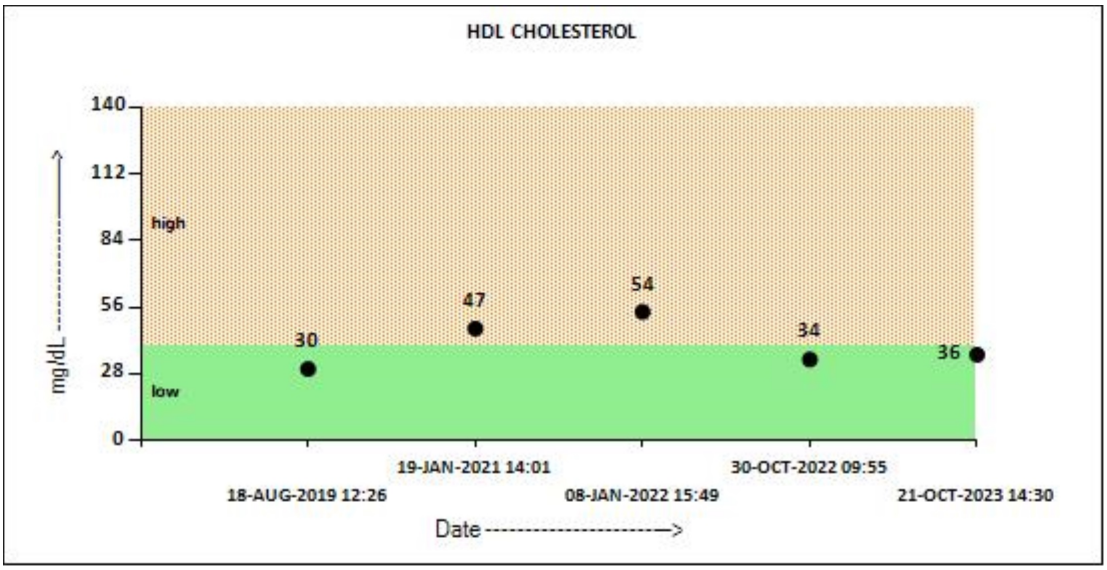

KIDNEY FUNCTION TEST

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BLOOD UREA NITROGEN METHOD : UREASE -GLDH	12	6 - 20	mg/dL
CREATININE METHOD : PICRATE / NAOH / JAFFE	1.06	0.90 - 1.30	mg/dL
BUN/CREAT RATIO METHOD : CALCULATED PARAMETER	11.32	5.00 - 15.00	
URIC ACID METHOD : URICASE UV	7.0	3.5 - 7.2	mg/dL
TOTAL PROTEIN METHOD : BIURET	6.9	6.4 - 8.2	g/dL
ALBUMIN METHOD : BCG DYE BINDING METHOD	3.9	3.4 - 5.0	g/dL
GLOBULIN METHOD : CALCULATED PARAMETER	3.0	2.0 - 4.1	g/dL
CALCIUM METHOD : O-CRESOLPHTHALEIN COMPLEXONE	8.5	8.5 - 10.1	mg/dL
SODIUM, SERUM METHOD : ION SELECTIVE ELECTRODE TECHNOLOGY	139	136 - 145	mmol/L
POTASSIUM, SERUM METHOD : ION SELECTIVE ELECTRODE TECHNOLOGY	4.04	3.50 - 5.10	mmol/L
CHLORIDE, SERUM METHOD : ION SELECTIVE ELECTRODE TECHNOLOGY	103	98 - 107	mmol/L

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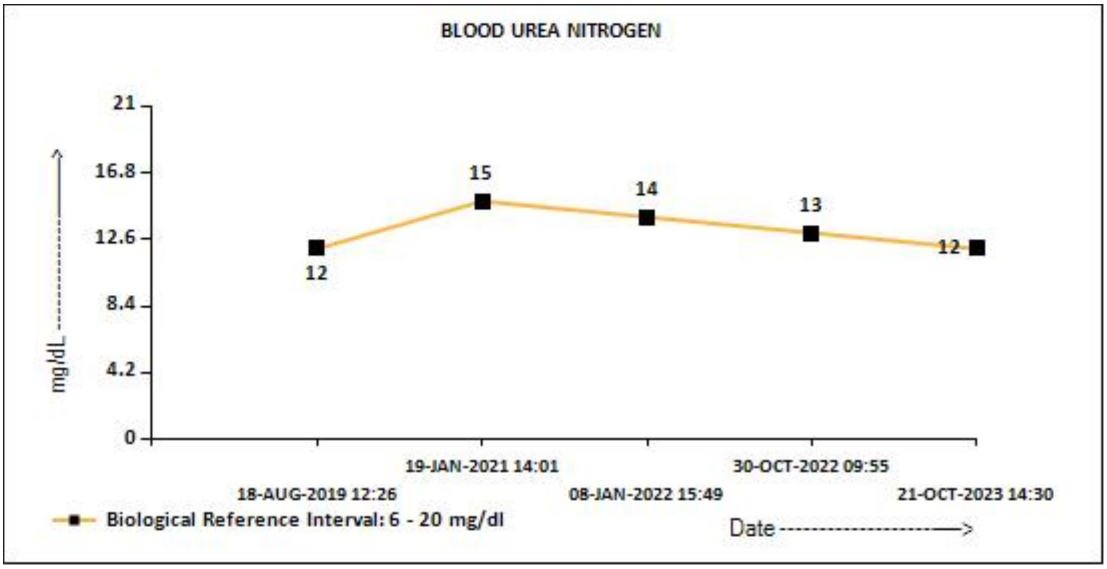

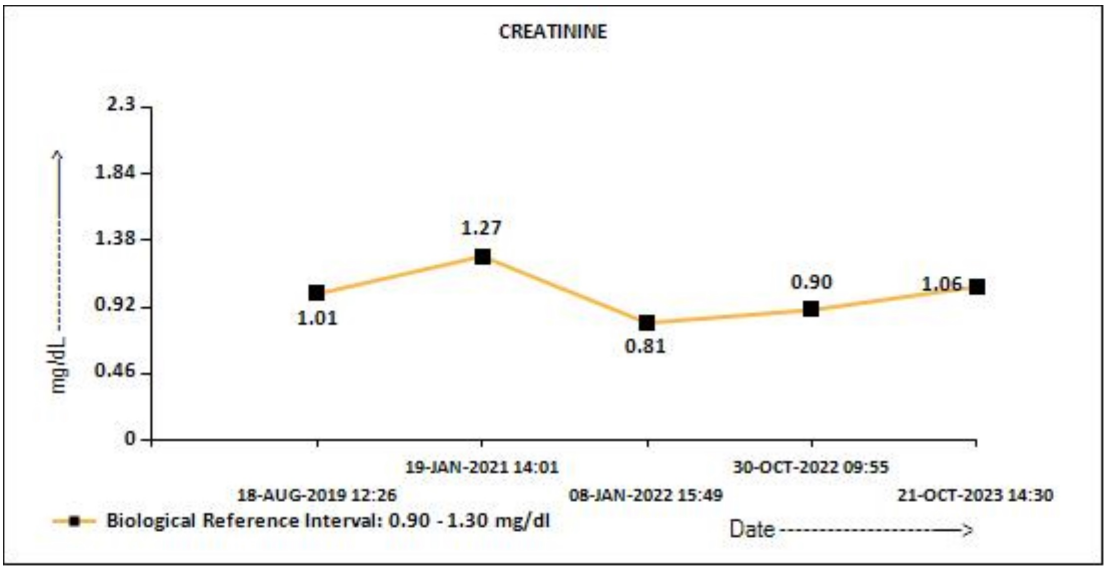

MAGNESIUM, SERUM

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PREETPAL SINGH S/O S.AMRIK SINGH AMRITSAR Amritsar 143001	ACCESSION NO : **0202WJ007460** PATIENT ID : PREEM19027978A CLIENT PATIENT ID: ABHA NO :	AGE/SEX :44 Years Male DRAWN :21/10/2023 12:36:27 RECEIVED :21/10/2023 12:39:49 REPORTED :21/10/2023 14:56:46

Test Report Status **Final**	Results	Biological Reference Interval	Units
MAGNESIUM, SERUM	1.8	1.8 - 2.4	mg/dL

Interpretation(s)
GLUCOSE FASTING,FLUORIDE PLASMA-**TEST DESCRIPTION**
Normally, the glucose concentration in extracellular fluid is closely regulated so that a source of energy is readily available to tissues and sothat no glucose is excreted in the urine.
Increased in:Diabetes mellitus, Cushing' s syndrome (10 – 15%), chronic pancreatitis (30%). Drugs:corticosteroids,phenytoin, estrogen, thiazides.
Decreased in :Pancreatic islet cell disease with increased insulin,insulinoma,adrenocortical insufficiency,hypopituitarism,diffuse liver disease, malignancy(adrenocortical,stomach,fibrosarcoma),infant of a diabetic mother,enzyme deficiency diseases(e.g.galactosemia),Drugs-insulin,ethanol,propranolol sulfonylureas,tolbutamide,and other oral hypoglycemic agents.
NOTE: While random serum glucose levels correlate with home glucose monitoring results (weekly mean capillary glucose values),there is wide fluctuation within individuals.Thus, glycosylated hemoglobin(HbA1c) levels are favored to monitor glycemic control.
High fasting glucose level in comparison to post prandial glucose level may be seen due to effect of Oral Hypoglycaemics & Insulin treatment,Renal Glyosuria,Glycaemic index & response to food consumed,Alimentary Hypoglycemia,Increased insulin response & sensitivity etc.
LIVER FUNCTION PROFILE, SERUM-
Bilirubin is a yellowish pigment found in bile and is a breakdown product of normal heme catabolism. Bilirubin is excreted in bile and urine, and elevated levels may give yellow discoloration in jaundice.**Elevated levels** results from increased bilirubin production (eg, hemolysis and ineffective erythropoiesis), decreased bilirubin excretion (eg, obstruction and hepatitis), and abnormal bilirubin metabolism (eg, hereditary and neonatal jaundice). Conjugated (direct) bilirubin is elevated more than unconjugated (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
MAGNESIUM, SERUM-Description- Magnesium is primariliy an intracellular ion associated with GI absorption and renal excretion. Second most abundant ion in bone.It functions as co-factor in numerous enzymes e.g. ATPase . 65-70% of Mg is in ionized state and nearly 35% is protein bound.

Interpretation-
Increased in- Dehydration, Tissue trauma, Renal failure, Hypothyroidism, excessive intake of antacid.
Decrease in- Chronic diarrhea, Enteric fistula, Starvation, Chronic alcoholism, Total parenteral Nutrition, Diuretics.

Note- Hypomagnesemia is associated with weakness, tetany, disorientation and somnolence

Limitation-
• Hemolysis yields elevated levels of Mg being an intracellular ion.
• Serum magnesium levels may remain normal even when total body stores of magnesium are depleted upto 20%

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Tel : 9111591115, Fax : CIN - U74899PB1995PLC045956

Patient Ref. No. 775000005180288

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SPECIALISED CHEMISTRY - ANEMIA

COMPLETE CARE TOTAL WITH SMART REPORT

SERUM IRON AND TIBC STUDIES

IRON	73	65 - 175	µg/dL
METHOD : CHROMOPHORE FERENE / ASCORBIC ACID			
TOTAL IRON BINDING CAPACITY	321	250 - 450	µg/dL
METHOD : TRANSFERRIN / FERENE			
% SATURATION	23	13 - 45	%
METHOD : CALCULATED PARAMETER			

Interpretation(s)
SERUM IRON AND TIBC STUDIES-Total iron binding capacity (TIBC) measures the blood's capacity to bind iron with transferrin and thus is an indirect way of assessing transferrin level.
Taken together with serum iron and percent transferrin saturation this test is performed when they is a concern about anemia, iron deficiency or iron deficiency anemia. However, because the liver produces transferrin, alterations in liver function (such as cirrhosis, hepatitis, or liver failure) must be considered when performing this test.
Increased in:
- iron deficiency
- acute and chronic blood loss
- acute liver damage
- progesterone birth control pills

Decreased in:
- hemochromatosis
- cirrhosis of the liver
- thalassemia
- anemias of infection and chronic diseases
- nephrosis
- hyperthyroidism

The percent Transferrin saturation = Serum Iron/TIBC x 100
Unsaturated Binding Capacity (UIBC)=TIBC - Serum Iron.
Limitations: Estrogens and oral contraceptives increase TIBC and Asparaginase, chloramphenicol, corticotropin, cortisone and testosterone decrease the TIBC level.

Reference:
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, 563, 1314-1315.
2. Wallach's Interpretation of Diagnostic tests, 9th Edition, Ed Mary A Williamson and L Michael Snyder. Pub Lippincott Williams and Wilkins, 2011, 234-235.

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CLINICAL PATH - URINALYSIS

COMPLETE CARE TOTAL WITH SMART REPORT

PHYSICAL EXAMINATION, URINE			
COLOR	PALE YELLOW		
APPEARANCE	CLEAR		
CHEMICAL EXAMINATION, URINE			
PH	6.0	4.5 - 7.5	
SPECIFIC GRAVITY	1.030	1.005 - 1.030	
PROTEIN	NOT DETECTED	NEGATIVE	
GLUCOSE	NOT DETECTED	NEGATIVE	
KETONES	NOT DETECTED	NOT DETECTED	
BLOOD	NOT DETECTED	NEGATIVE	
BILIRUBIN	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)	2-3	0-5	/HPF
EPITHELIAL CELLS	NOT DETECTED	0-5	/HPF
CASTS	NOT DETECTED		
CRYSTALS	NOT DETECTED		
BACTERIA	NOT DETECTED	NOT DETECTED	
YEAST	NOT DETECTED	NOT DETECTED	

Comments

URINE MICROSCOPIC EXAMINATION PERFORMED ON DEPOSIT AFTER CENTRIFUGATION.

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SPECIALISED CHEMISTRY - HORMONE

COMPLETE CARE TOTAL WITH SMART REPORT

THYROID PANEL, SERUM

T3	108.85	60.0 - 181.0	ng/dL
METHOD : CHEMILUMINESCENCE			
T4	7.20	4.5 - 10.9	µg/dL
METHOD : CHEMILUMINESCENCE			
TSH (ULTRASENSITIVE)	2.542	0.550 - 4.780	µIU/mL
METHOD : CHEMILUMINESCENCE			

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SPECIALISED CHEMISTRY - VITAMIN

COMPLETE CARE TOTAL WITH SMART REPORT

25 - HYDROXYVITAMIN D(VITAMIN D TOTAL), SERUM

25 - HYDROXYVITAMIN D	**25.25 Low**	Deficiency: < 20.0 Insufficiency: 20.0 - 30.0 Sufficiency: 30.0 - 100.0 Toxicity > 100.0	ng/mL

METHOD : CHEMILUMINESCENCE

VITAMIN B12(CYANOCOBALAMINE), SERUM

VITAMIN B12	342.0	211.0 - 911.0	pg/mL

METHOD : CHEMILUMINESCENCE

Interpretation(s)

25 - HYDROXYVITAMIN D(VITAMIN D TOTAL), SERUM-**Test description**

Vitamin D has anti-inflammatory and immune-modulating properties and it works towards the bones, teeth, intestines, immune system, pancreas, muscles and brain. It helps to maintain normal calcium and phosphate levels.Vitamin D is a fat-soluble vitamin. Also called as "Sunshine Vitamin".Two main forms as Cholecalciferol (vitamin D3) which is synthesized in skin from 7-dehydrocholesterol in response to sunlight (Type B UV) exposure & Ergocalciferol (vitamin D2) present mainly in dietary sources.

Vit D25(OH)D deficiency is seen due to poor or inadequate sunlight exposure, Nutritional or dietary deficiency or fat malabsorption, Severe Hepatocellular disease, Secondary hyperparathyroidism, Hypocalcemia tetany which can cause involuntary contraction of muscles, leading to cramps and spasms, Rickets in children, Osteomalacia in adults- due to vitamin D deficiency mainly, Older adults- osteoporosis. (Increased risk of bone fractures)due to long-term effect of calcium and/or vitamin D deficiency, Other conditions that are precipitated by Vit D deficiency included increased cardiovascular risk, low immunity & chronic renal failure.

Elevated levels may be seen in patients taking supplements(hence recommended to repeat after 3 months for estimation of accurate levels), Vitamin D intoxication, sarcoidosis and malignancies containing non regulated 1-alpha hydroxylase in the lesion.

Recommendations

1.To prevent biotin interference the patient should be atleast 8 hours fasting before submitting the sample 2.25(OH)D is the analyte of choice for determination of the Vitamin D status as it is the major storage & active form of Vitamin D and has longer half-life. 3. Kidney Disease Outcomes Quality Initiatives (KDOQI) and Kidney Disease Improving Global Outcomes (KDIGO) recommend activated vitamin D testing for CKD patients.

Note-Our Vitamin D assays is standardized to be in alignment with the ID-LC/MS/MS 25(OH)vitamin D Reference Method Procedure (RMP), the reference procedure for the Vitamin D Standardization Program (VDSP). The VDSP, a collaboration of the National Institutes of Health Office of Dietary Supplements, National Institute of Technology and Standards, Centers for Disease Control and Ghent University, is an initiative to standardize 25(OH)vitamin D measurement across methods.

Reference:

1.Wallach Interpretation of diagnostic test, 10th edition.

VITAMIN B12(CYANOCOBALAMINE), SERUM-**Test description**

1.Measures the amount of Vitamin B12/ Cyanocobalamin or Methyl cobalamin in blood.2. Done in Anemic conditions like Megaloblastic anemia, pernicious anemia, dietary folate deficiencies,3.Workup of neuropathies especially due to diabetes.4.Nerve health and it is monitored in treatment of nerve damage.5.Important vitamin for women of childbearing age and for older people.

1.Part of water-soluble B complex of vitamins. 2. It is essential in DNA synthesis, hematopoiesis & CNS integrity.3.Source for B12 is dietary foods like milk, yoghurt, eggs, meat, fortified cereals, bread. 4.Absorption depends on the HCl secreted by the stomach and occurs in intestines. 5. It is part of enterohepatic circulation, hence excreted in feces(approx. 0.1% per day)

Test interpretation

Higher than normal levels are in patients on Vitamin supplements or patients with COPD, CRF, Diabetes, Liver cell damage, Obesity, Polycythemia.

Decreased levels seen in

Inflammatory bowel disease, Pernicious anemia - genetic deficiency of intrinsic factor - necessary for Vit B12 absorption, Strict vegetarianslead to sub-clinical B12 deficiency- high among elderly patients, Malabsorption due to gastrectomy, smoking, pregnancy, multiple myeloma & hemodialysis, Alcohol & drugs like amino salicylic acid, anticonvulsants, cholestyramine, cimetidine, Hyperthyroidism (High levels of thyroid), Seen in mothers of children with (NTD) Neural tube defects- hence fortification and

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supplements are advised in expecting mothers
Recommendations-1.To prevent biotin interference the patient should be atleast 8 hours fasting before submitting the sample. 2. Vit B12 and Folic acid evaluated together in macrocytic anemias to avoid methyl folate trap. Carmel's composite criteria for inadequate Vit B12 status: Serum vitamin B12 < 148 pmol/L, or 148–258 pmol/L and MMA > 0.30µmol/L, or tHcy > 13 nmol/L (females) and >15 nmol/L (males).
Associated Test-Holo-TC: Marker of vitamin B12 status -specificity and sensitivity better than serum vitamin B12, hence recommended in boderline and deficient cases for confirmation.
References-O''''''''''''''Leary F, Samman S. Vitamin B12 in health and disease. Nutrients. 2010 Mar 2(3):299-316.

****End Of Report****

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

CONDITIONS OF LABORATORY TESTING & REPORTING

1. It is presumed that the test sample belongs to the patient named or identified in the test requisition form.
2. All tests are performed and reported as per the turnaround time stated in the AGILUS Directory of Services.
3. Result delays could occur due to unforeseen circumstances such as non-availability of kits / equipment breakdown / natural calamities / technical downtime or any other unforeseen event.
4. A requested test might not be performed if:
   i. Specimen received is insufficient or inappropriate
   ii. Specimen quality is unsatisfactory
   iii. Incorrect specimen type
   iv. Discrepancy between identification on specimen container label and test requisition form
5. AGILUS Diagnostics confirms that all tests have been performed or assayed with highest quality standards, clinical safety & technical integrity.
6. Laboratory results should not be interpreted in isolation; it must be correlated with clinical information and be interpreted by registered medical practitioners only to determine final diagnosis.
7. Test results may vary based on time of collection, physiological condition of the patient, current medication or nutritional and dietary changes. Please consult your doctor or call us for any clarification.
8. Test results cannot be used for Medico legal purposes.
9. In case of queries please call customer care (91115 91115) within 48 hours of the report.

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