

CODE/NAME & ADDRESS: C000139306 ACCESSION NO: 0340XF000040 AGE/SEX :60 Years

MED ACTIVE

PLOT NO. GM-15, NEAR BSNL CHOWK, PHASE

II,CHHEND COLONY ROURKELA 769015 7061912526

PATIENT ID : SANAM140419650

CLIENT PATIENT ID: ABHA NO

DRAWN :02/06/2024 12:29:55 RECEIVED: 02/06/2024 17:53:17 REPORTED :02/06/2024 20:51:28

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

BIOCHEMISTRY

AGILUS SCREEN 1

GLUCOSE FASTING, FLUORIDE PLASMA

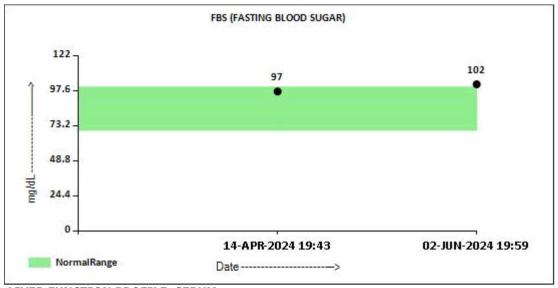
FBS (FASTING BLOOD SUGAR)

102 High

(Normal <100,Impaired fastimg/dL glucose:100 to 125, Diabetes mellitus:>=126(on more than 1 occasion)(ADA guidelines

2024)

METHOD: GOD-POD METHOD



LIVER FUNCTION PROFILE, SERUM

BILIRUBIN, TOTAL	0.30	0.3 - 1.2	mg/dL
METHOD: DIAZO METHOD BILIRUBIN, DIRECT	0.17	0.00 - 0.40	mg/dL
METHOD : DIAZO METHOD BILIRUBIN, INDIRECT	0.13	0.1 - 1.0	mg/dL
METHOD: CALCULATED PARAMETER TOTAL PROTEIN	7.9	6.4 - 8.3	g/dL
ALBUMIN	4.3	3.2 - 4.6	g/dL

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Page 1 Of 11





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GLOBULIN	3.6 High	2.3 - 3.5	g/dL
ALBUMIN/GLOBULIN RATIO	1.2	1.0 - 2.1	RATIO
ASPARTATE AMINOTRANSFERASE(AST/SGOT)	20	0.0 - 35.0	U/L
ALANINE AMINOTRANSFERASE (ALT/SGPT)	25	0.0 - 45.0	U/L
ALKALINE PHOSPHATASE	55 Low	56 - 119	U/L
GAMMA GLUTAMYL TRANSFERASE (GGT)	33	0.0 - 55.0	U/L
LACTATE DEHYDROGENASE	321	225 - 450	U/L
KIDNEY FUNCTION TEST, SERUM			
BLOOD UREA NITROGEN METHOD: UREASE -GLDH	12	8.0 - 26.0	mg/dL
CREATININE METHOD: JAFFE KINETIC METHOD	0.95	0.7 - 1.3	mg/dL
BUN/CREAT RATIO METHOD: CALCULATED PARAMETER	12.63	5.0 - 15.0	
URIC ACID METHOD: URICASE	4.7	3.5 - 7.2	mg/dL
TOTAL PROTEIN METHOD: BIURET	7.9	6.4 - 8.3	g/dL
ALBUMIN METHOD: BCG	4.3	3.2 - 4.6	g/dL
GLOBULIN	3.6 High	2.3 - 3.5	g/dL
CALCIUM METHOD: ARSENAZO III	9.8	8.6 - 10.2	mg/dL
SODIUM, SERUM METHOD: ISE INDIRECT	141.6	136.0 - 146.0	mmol/L
POTASSIUM, SERUM METHOD: ISE INDIRECT	3.62	3.50 - 5.10	mmol/L
CHLORIDE, SERUM METHOD: ISE INDIRECT	103.0	98.0 - 106.0	mmol/L
LIPID PROFILE WITH CALCULATED LDL, SERUM	1		
CHOLESTEROL, TOTAL	124	< 200 Desirable 200 - 239 Borderline	mg/dL

METHOD: CHOD-POD

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> or = 240 High



Page 2 Of 11

View Report



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Test Report Status <u>Final</u>	Results	Biological Reference Interva	l Units
TRIGLYCERIDES	97	< 161 Normal 161 - 199 High 200 - 499 Hypertriglyceride > or = 500 Very High	mg/dL mia
METHOD : GPO - POD METHOD HDL CHOLESTEROL METHOD : DIRECT	35 Low	35.3 - 79.5	mg/dL
CHOLESTEROL LDL	70	< 100 Optimal 100 - 129 Near or above optimal 130 - 159 Borderline High 160 - 189 High >/= 190 Very High	mg/dL
NON HDL CHOLESTEROL	89	Desirable: Less than 130 Above Desirable: 130 - 159 Borderline High: 160 - 189 High: 190 - 219 Very high: > or = 220	mg/dL
VERY LOW DENSITY LIPOPROTEIN	19.4	< 30.0	mg/dL
CHOL/HDL RATIO	3.5	3.30 - 4.40	
LDL/HDL RATIO	2	0.50 - 3.00	



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Page 3 Of 11

View Details

View Report



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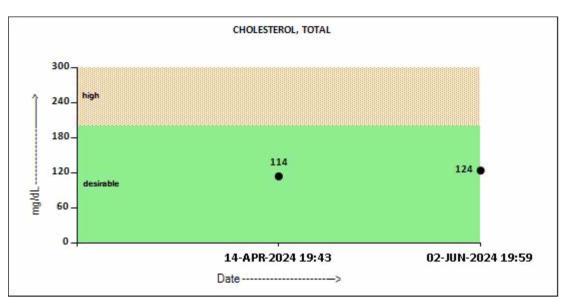
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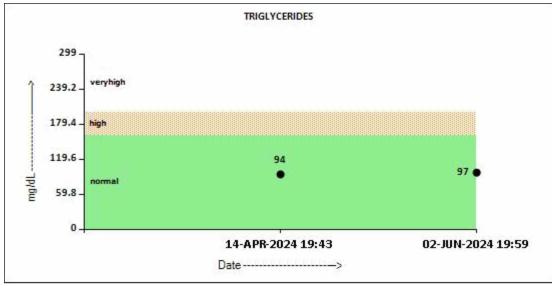
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Biological Reference Interval **Test Report Status** Results Units **Final**







Odisha, India

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Page 4 Of 11





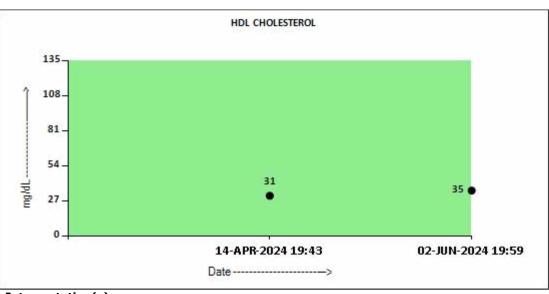
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Interpretation(s)

Serum lipid profile is measured for cardiovascular risk prediction. Lipid Association of India recommends LDL-C as primary target and Non HDL-C as co-primary treatment target.

Risk Stratification for ASCVD (Atherosclerotic cardiovascular disease) by Lipid Association of India

Risk Category					
Extreme risk group	A.CAD with > 1 feature of high risk group				
	B. CAD with > 1 feature of Very high risk g	group or recurrent ACS (within 1 year) despite LDL-C < or =			
	50 mg/dl or polyvascular disease				
Very High Risk	1. Established ASCVD 2. Diabetes with 2 1	najor risk factors or evidence of end organ damage 3.			
	Familial Homozygous Hypercholesterolemi	a			
High Risk	1. Three major ASCVD risk factors. 2. Dia	betes with 1 major risk factor or no evidence of end organ			
	damage. 3. CKD stage 3B or 4. 4. LDL >190 mg/dl 5. Extreme of a single risk factor. 6. Coronary				
	Artery Calcium - CAC >300 AU. 7. Lipoprotein a >/= 50mg/dl 8. Non stenotic carotid plaque				
Moderate Risk	2 major ASCVD risk factors				
Low Risk	0-1 major ASCVD risk factors				
Major ASCVD (Atherosclerotic cardiovascular disease) Risk Factors					
1. Age $>$ or = 45 years in males and $>$ or = 55 years in females 3. Current Cigarette smoking or tobacco use					
2. Family history of premature ASCVD 4. High blood pressure					
5. Low HDL					
T	1 4 2 1 22 2 4 1 1 1 1 1 1	11 7 17 1 0000			

Newer treatment goals and statin initiation thresholds based on the risk categories proposed by LAI in 2020.

Treatment Goals

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Risk Group



Consider Drug Therapy



Page 5 Of 11

View Details

View Report





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	LDL-C (mg/dl)	Non-HDL (mg/dl)	LDL-C (mg/dl)	Non-HDL (mg/dl)
Extreme Risk Group Category A	<50 (Optional goal < OR = 30)	< 80 (Optional goal <or 60)<="" =="" td=""><td>>OR = 50</td><td>>OR = 80</td></or>	>OR = 50	>OR = 80
Extreme Risk Group Category B	<or 30<="" =="" td=""><td><or 60<="" =="" td=""><td>> 30</td><td>>60</td></or></td></or>	<or 60<="" =="" td=""><td>> 30</td><td>>60</td></or>	> 30	>60
Very High Risk	<50	<80	>OR= 50	>OR= 80
High Risk	<70	<100	>OR= 70	>OR= 100
Moderate Risk	<100	<130	>OR= 100	>OR= 130
Low Risk	<100	<130	>OR= 130*	>OR= 160

^{*}After an adequate non-pharmacological intervention for at least 3 months.

References: Management of Dyslipidaemia for the Prevention of Stroke: Clinical Practice Recommendations from the Lipid Association of India. Current Vascular Pharmacology, 2022, 20, 134-155.

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

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 nse 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 wher 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

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Page 6 Of 11

View Report





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CLINICAL PATH - URINALYSIS

AGILUS SCREEN 1

PHYSICAL EXAMINATION, URINE

COLOR PALE YELLOW

APPEARANCE CLEAR

CHEMICAL EXAMINATION, URINE

PH	6.0	4.7 - 7.5
SPECIFIC GRAVITY	1.030	1.003 - 1.035
PROTEIN	NOT DETECTED	NOT DETECTED
GLUCOSE	NOT DETECTED	NOT DETECTED
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	1-2	0-5	/HPF
CASTS	NOT DETECTED		

NOT DETECTED **CRYSTALS**

BACTERIA NOT DETECTED NOT DETECTED YEAST **NOT DETECTED** NOT DETECTED

Interpretation(s)

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Page 7 Of 11





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The following table describes the probable conditions, in which the analytes are present in urine

Presence of	Conditions			
Proteins	Inflammation or immune illnesses			
Pus (White Blood Cells)	Urinary tract infection, urinary tract or kidney stone, tumors or any kind of kidney impairment			
Glucose	Diabetes or kidney disease			
Ketones	Diabetic ketoacidosis (DKA), starvation or thirst			
Urobilinogen	Liver disease such as hepatitis or cirrhosis			
Blood	Renal or genital disorders/trauma			
Bilirubin	Liver disease			
Erythrocytes	Urological diseases (e.g. kidney and bladder cancer, urolithiasis), urinary tract infection and glomerular diseases			
Leukocytes	Urinary tract infection, glomerulonephritis, interstitial nephritis either acute or chronic, polycystic kidney disease, urolithiasis, contamination by genital secretions			
Epithelial cells	Urolithiasis, bladder carcinoma or hydronephrosis, ureteric stents or bladder catheters for prolonged periods of time			
Granular Casts	Low intratubular pH, high urine osmolality and sodium concentration, interaction with Bence-Jones protein			
Hyaline casts	Physical stress, fever, dehydration, acute congestive heart failure, renal diseases			
Calcium oxalate	Metabolic stone disease, primary or secondary hyperoxaluria, intravenous infusion of large doses of vitamin C, the use of vasodilator naftidrofuryl oxalate or the gastrointestinal lipase inhibitor orlistat, ingestion of ethylene glycol or of star fruit (Averrhoa carambola) or its juice			
Uric acid	arthritis			
Bacteria	Urinary infectionwhen present in significant numbers & with pus cells.			
Trichomonas vaginalis	Vaginitis, cervicitis or salpingitis			

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Page 8 Of 11

View Details

View Penort



REF. DOCTOR: SELF **PATIENT NAME: SANATAN SAHOO**

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SPECIALISED CHEMISTRY - HORMONE

AGILUS SCREEN 1

THYROID PANEL, SERUM

69.0-215.0 T3 158.00 ng/dL

Pregnant women:

First trimester: 120-339 Bitmap Second trimester: 136-

Third trimester: 122-260

T4 6.96 5.2 - 12.7 µg/dL 8.720 High TSH (ULTRASENSITIVE) 0.3-4.5 IU/mL

> Bitmap Pregnant women: First trimester: 0.25-4.33 Bitmap Bitmap Second

trimester: 0.43-6.61 Third trimester: 0.38-6.22

Interpretation(s)

Triiodothyronine T3, Thyroxine T4, and Thyroid Stimulating Hormone TSH are thyroid hormones which affect almost every physiological process in the body, including growth, development, metabolism, body temperature, and heart rate.

Production of T3 and its prohormone thyroxine (T4) is activated by thyroid-stimulating hormone (TSH), which is released from the pituitary gland. Elevated concentrations of T3, and T4 in the blood inhibit the production of TSH.

Excessive secretion of thyroxine in the body is hyperthyroidism, and deficient secretion is called hypothyroidism.

In primary hypothyroidism, TSH levels are significantly elevated, while in secondary and tertiary hypothyroidism, TSH levels are low. Below mentioned are the guidelines for Pregnancy related reference ranges for Total T4, TSH & Total T3. Measurement of the serum TT3 level is a more sensitive test for the diagnosis of hyperthyroidism, and measurement of TT4 is more useful in the diagnosis of hypothyroidism. Most of the thyroid hormone in blood is bound to transport proteins. Only a very small fraction of the circulating hormone is free and biologically active. It is advisable to detect Free T3, Free T4 along with TSH, instead of testing for albumin bound Total T3, Total T4.

Sr. No.	TSH	Total T4	FT4	Total T3	Possible Conditions
1	High	Low	Low	Low	(1) Primary Hypothyroidism (2) Chronic autoimmune Thyroiditis (3)
					Post Thyroidectomy (4) Post Radio-Iodine treatment





Page 9 Of 11





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2	High	Normal	Normal	Normal	(1)Subclinical Hypothyroidism (2) Patient with insufficient thyroid
~	Thigh	1 tornur	TOTIM	1 (Ollina)	hormone replacement therapy (3) In cases of Autoimmune/Hashimoto
					thyroiditis (4). Isolated increase in TSH levels can be due to Subclinical
					inflammation, drugs like amphetamines, Iodine containing drug and
					dopamine antagonist e.g. domperidone and other physiological reasons.
3	Normal/Low	Low	Low	Low	(1) Secondary and Tertiary Hypothyroidism
4	Low	High	High	High	(1) Primary Hyperthyroidism (Graves Disease) (2) Multinodular Goitre
					(3)Toxic Nodular Goitre (4) Thyroiditis (5) Over treatment of thyroid
					hormone (6) Drug effect e.g. Glucocorticoids, dopamine, T4
					replacement therapy (7) First trimester of Pregnancy
5	Low	Normal	Normal	Normal	(1) Subclinical Hyperthyroidism
6	High	High	High	High	(1) TSH secreting pituitary adenoma (2) TRH secreting tumor
7	Low	Low	Low	Low	(1) Central Hypothyroidism (2) Euthyroid sick syndrome (3) Recent
					treatment for Hyperthyroidism
8	Normal/Low	Normal	Normal	High	(1) T3 thyrotoxicosis (2) Non-Thyroidal illness
9	Low	High	High	Normal	(1) T4 Ingestion (2) Thyroiditis (3) Interfering Anti TPO antibodies

REF: 1. TIETZ Fundamentals of Clinical chemistry 2. Guidlines of the American Thyroid association during pregnancy and Postpartum, 2011. NOTE: It is advisable to detect Free T3, FreeT4 along with TSH, instead of testing for albumin bound Total T3, Total T4.TSH is not affected by variation in thyroid - binding protein. TSH has a diurnal rhythm, with peaks at 2:00 - 4:00 a.m. And troughs at 5:00 - 6:00 p.m. With ultradian variations.

> **End Of Report** Please visit www.agilusdiagnostics.com for related Test Information for this accession

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Page 10 Of 11





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- 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.
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 - 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

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- 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.
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Agilus Diagnostics Ltd

Fortis Hospital, Sector 62, Phase VIII, Mohali 160062

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Page 11 Of 11

View Details

View Repor

