PATIENT NAME : NISHI GUPTA | **REF. DOCTOR :** SELF

CODE/NAME & ADDRESS :C000049066
AGILUS DIAGNOSTICS LIMITED-WEL WALK-IN-
AAKRITI LABS PVT LTD. A-430, AGRASEN MARG
JAIPUR 302017
9314660100

ACCESSION NO : **0251XA002187**
PATIENT ID : NISHF231068251
CLIENT PATIENT ID: 012401270013
ABHA NO :

AGE/SEX : 56 Years Female
DRAWN : 27/01/2024 08:54:00
RECEIVED : 27/01/2024 09:31:19
REPORTED : 28/01/2024 09:46:28

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

HAEMATOLOGY - CBC

MEDI WHEEL FULL BODY HEALTH CHECKUP ABOVE 40FEMALE

BLOOD COUNTS,EDTA WHOLE BLOOD

HEMOGLOBIN (HB) METHOD : CYANIDE FREE DETERMINATION	14.0	12.0 - 15.0	g/dL
RED BLOOD CELL (RBC) COUNT METHOD : ELECTRICAL IMPEDANCE	**5.48 High**	3.8 - 4.8	mil/µL
WHITE BLOOD CELL (WBC) COUNT METHOD : ELECTRICAL IMPEDANCE	6.50	4.0 - 10.0	thou/µL
PLATELET COUNT METHOD : ELECTRONIC IMPEDANCE	189	150 - 410	thou/µL

RBC AND PLATELET INDICES

HEMATOCRIT (PCV) METHOD : CALCULATED PARAMETER	44.6	36 - 46	%
MEAN CORPUSCULAR VOLUME (MCV) METHOD : CALCULATED PARAMETER	**81.0 Low**	83 - 101	fL
MEAN CORPUSCULAR HEMOGLOBIN (MCH) METHOD : CALCULATED PARAMETER	**25.6 Low**	27.0 - 32.0	pg
MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION (MCHC) METHOD : CALCULATED PARAMETER	**31.4 Low**	31.5 - 34.5	g/dL
RED CELL DISTRIBUTION WIDTH (RDW) METHOD : CALCULATED PARAMETER	**16.3 High**	11.6 - 14.0	%
MENTZER INDEX	14.8		
MEAN PLATELET VOLUME (MPV) METHOD : CALCULATED PARAMETER	10.6	6.8 - 10.9	fL

WBC DIFFERENTIAL COUNT

NEUTROPHILS METHOD : IMPEDANCE WITH HYDRO FOCUS AND MICROSCOPY	58	40 - 80	%
LYMPHOCYTES METHOD : IMPEDANCE WITH HYDRO FOCUS AND MICROSCOPY	35	20 - 40	%
MONOCYTES	02	2 - 10	%

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Patient Ref. No. 775000006205912

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METHOD : IMPEDANCE WITH HYDRO FOCUS AND MICROSCOPY EOSINOPHILS	05	1 - 6	%
METHOD : IMPEDANCE WITH HYDRO FOCUS AND MICROSCOPY BASOPHILS	00	0 - 2	%
METHOD : IMPEDANCE WITH HYDRO FOCUS AND MICROSCOPY ABSOLUTE NEUTROPHIL COUNT	3.77	2.0 - 7.0	thou/µL
METHOD : CALCULATED PARAMETER ABSOLUTE LYMPHOCYTE COUNT	2.28	1.0 - 3.0	thou/µL
METHOD : CALCULATED PARAMETER ABSOLUTE MONOCYTE COUNT	**0.13 Low**	0.2 - 1.0	thou/µL
METHOD : CALCULATED PARAMETER ABSOLUTE EOSINOPHIL COUNT	0.32	0.02 - 0.50	thou/µL
METHOD : CALCULATED PARAMETER ABSOLUTE BASOPHIL COUNT	**0 Low**	0.02 - 0.10	thou/µL
NEUTROPHIL LYMPHOCYTE RATIO (NLR)	1.7		

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

MEDI WHEEL FULL BODY HEALTH CHECKUP ABOVE 40FEMALE

GLYCOSYLATED HEMOGLOBIN(HBA1C), EDTA WHOLE BLOOD

HBA1C	5.6	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)	114.0	< 116.0	mg/dL
METHOD : CALCULATED PARAMETER			

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MEDI WHEEL FULL BODY HEALTH CHECKUP ABOVE 40FEMALE

ERYTHROCYTE SEDIMENTATION RATE (ESR),EDTA BLOOD

E.S.R	19	0 - 20	mm at 1 hr

METHOD : AUTOMATED (PHOTOMETRICAL CAPILLARY STOPPED FLOW KINETIC ANALYSIS)"

Interpretation(s)
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

ERYTHROCYTE SEDIMENTATION RATE (ESR),EDTA 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 ESR 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: Polycythemia 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.

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IMMUNOHAEMATOLOGY

MEDI WHEEL FULL BODY HEALTH CHECKUP ABOVE 40FEMALE

ABO GROUP & RH TYPE, EDTA WHOLE BLOOD

ABO GROUP METHOD : TUBE AGGLUTINATION	TYPE B		
RH TYPE METHOD : TUBE AGGLUTINATION	POSITIVE		

Interpretation(s)
ABO GROUP & RH TYPE, EDTA WHOLE BLOOD-Blood group is identified byantigens and antibodies present in the blood. Antigens are protein molecules found on the surface of red blood cells. Antibodies are found in plasma. To determine blood group, red cells are mixed with different antibody solutions to give A,B,O or AB.

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

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

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BIOCHEMISTRY

MEDI WHEEL FULL BODY HEALTH CHECKUP ABOVE 40FEMALE

GLUCOSE FASTING,FLUORIDE PLASMA

FBS (FASTING BLOOD SUGAR) METHOD : GLUCOSE OXIDASE	95	74 - 99	mg/dL

GLUCOSE, POST-PRANDIAL, PLASMA

PPBS(POST PRANDIAL BLOOD SUGAR) METHOD : GLUCOSE OXIDASE	115	70 - 140	mg/dL

LIPID PROFILE WITH CALCULATED LDL

CHOLESTEROL, TOTAL METHOD : CHOLESTEROL OXIDASE	183	< 200 Desirable 200 - 239 Borderline High >/= 240 High	mg/dL
TRIGLYCERIDES METHOD : LIPASE/GPO-PAP NO CORRECTION	84	< 150 Normal 150 - 199 Borderline High 200 - 499 High >/=500 Very High	mg/dL
HDL CHOLESTEROL METHOD : DIRECT CLEARANCE METHOD	42	< 40 Low >/=60 High	mg/dL
CHOLESTEROL LDL	**124 High**	< 100 Optimal 100 - 129 Near optimal/ above optimal 130 - 159 Borderline High 160 - 189 High >/= 190 Very High	mg/dL
NON HDL CHOLESTEROL METHOD : CALCULATED PARAMETER	**141 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	16.8	</= 30.0	mg/dL
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	
LDL/HDL RATIO	3.0	0.5 - 3.0 Desirable/Low Risk 3.1 - 6.0 Borderline/Moderate Risk >6.0 High Risk	

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 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 major risk factors or evidence of end organ damage 3. Familial Homozygous Hypercholesterolemia
High Risk	1. Three major ASCVD risk factors. 2. Diabetes 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	

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

Risk Group	**Treatment Goals**		**Consider Drug Therapy**	
	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)	>OR = 50	>OR = 80

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Extreme Risk Group Category B	<OR = 30	<OR = 60	> 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.

LIVER FUNCTION PROFILE, SERUM

BILIRUBIN, TOTAL	0.55	0 - 1	mg/dL
METHOD : DIAZO WITH SULPHANILIC ACID			
BILIRUBIN, DIRECT	0.19	0.00 - 0.25	mg/dL
METHOD : DIAZO WITH SULPHANILIC ACID			
BILIRUBIN, INDIRECT	0.36	0.1 - 1.0	mg/dL
METHOD : CALCULATED PARAMETER			
TOTAL PROTEIN	7.9	6.4 - 8.2	g/dL
METHOD : BIURET REACTION, END POINT			
ALBUMIN	**4.5 High**	3.8 - 4.4	g/dL
METHOD : BROMOCRESOL GREEN			
GLOBULIN	3.4	2.0 - 4.1	g/dL
METHOD : CALCULATED PARAMETER			
ALBUMIN/GLOBULIN RATIO	1.3	1.0 - 2.1	RATIO
METHOD : CALCULATED PARAMETER			
ASPARTATE AMINOTRANSFERASE(AST/SGOT)	**37 High**	0 - 31	U/L
METHOD : TRIS BUFFER NO P5P IFCC / SFBC 37° C			
ALANINE AMINOTRANSFERASE (ALT/SGPT)	**37 High**	0 - 31	U/L
METHOD : TRIS BUFFER NO P5P IFCC / SFBC 37° C			
ALKALINE PHOSPHATASE	96	39 - 117	U/L
METHOD : AMP OPTIMISED TO IFCC 37° C			
GAMMA GLUTAMYL TRANSFERASE (GGT)	23	7 - 32	U/L
METHOD : GAMMA GLUTAMYL-3 CARBOXY-4 NITROANILIDE (IFCC) 37° C			
LACTATE DEHYDROGENASE	343	230 - 460	U/L

BLOOD UREA NITROGEN (BUN), SERUM

BLOOD UREA NITROGEN	12	5.0 - 18.0	mg/dL
METHOD : UREASE KINETIC			

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CREATININE, SERUM			
CREATININE METHOD : ALKALINE PICRATE NO DEPROTEINIZATION	0.87	0.6 - 1.2	mg/dL
BUN/CREAT RATIO			
BUN/CREAT RATIO METHOD : CALCULATED PARAMETER	13.79		
URIC ACID, SERUM			
URIC ACID METHOD : URICASE PEROXIDASE WITH ASCORBATE OXIDASE	5.4	2.4 - 5.7	mg/dL
TOTAL PROTEIN, SERUM			
TOTAL PROTEIN METHOD : BIURET REACTION, END POINT	7.9	6.4 - 8.3	g/dL
ALBUMIN, SERUM			
ALBUMIN METHOD : BROMOCRESOL GREEN	**4.5 High**	3.8 - 4.4	g/dL
GLOBULIN			
GLOBULIN	3.4	2.0 - 4.1	g/dL
ELECTROLYTES (NA/K/CL), SERUM			

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SODIUM, SERUM METHOD : ION-SELECTIVE ELECTRODE	142.2	137 - 145	mmol/L
POTASSIUM, SERUM METHOD : ION-SELECTIVE ELECTRODE	4.30	3.6 - 5.0	mmol/L
CHLORIDE, SERUM METHOD : ION-SELECTIVE ELECTRODE	103.4	98 - 107	mmol/L

Interpretation(s)

Sodium	Potassium	Chloride
Decreased in:CCF,cirrhosis, vomiting, diarrhea, excessive sweating, salt-losing nephropathy,adrenal insufficiency, nephrotic syndrome, water intoxication, SIADH. Drugs: thiazides, diuretics, ACE inhibitors, chlorpropamide,carbamazepine,anti depressants (SSRI), antipsychotics.	**Decreased in:** Low potassium intake,prolonged vomiting or diarrhea, RTA types I and II, hyperaldosteronism, Cushing's syndrome,osmotic diuresis (e.g., hyperglycemia),alkalosis, familial periodic paralysis,trauma (transient).Drugs: Adrenergic agents, diuretics.	**Decreased in:** Vomiting, diarrhea, renal failure combined with salt deprivation, over-treatment with diuretics, chronic respiratory acidosis, diabetic ketoacidosis, excessive sweating, SIADH, salt-losing nephropathy, porphyria, expansion of extracellular fluid volume, adrenalinsufficiency, hyperaldosteronism,metabolic alkalosis. Drugs: chronic laxative,corticosteroids, diuretics.
Increased in: Dehydration (excessivesweating, severe vomiting or diarrhea),diabetes *mellitus, diabetesinsipidus,* hyperaldosteronism, inadequate water intake. Drugs: steroids, licorice,oral contraceptives.	**Increased in:** Massive hemolysis, severe tissue damage, rhabdomyolysis, acidosis, dehydration,renal failure, *Addison' s disease, RTA type IV,* hyperkalemic familial periodic paralysis. Drugs: potassium salts, potassium- sparing diuretics,NSAIDs, beta-blockers, ACE inhibitors, high-dose trimethoprim-sulfamethoxazole.	**Increased in:** Renal failure, nephrotic syndrome, RTA,dehydration, overtreatment with *saline,hyperparathyroidism, diabetes* insipidus, metabolic acidosis from diarrhea (Loss of HCO3-), respiratory alkalosis,hyperadrenocorticism. Drugs: acetazolamide,androgens, hydrochlorothiazide,salicylates.
Interferences: Severe lipemia or hyperproteinemi, if sodium analysis involves a dilution step can cause spurious results. The serum sodium falls about 1.6 mEq/L for each 100 mg/dL increase in blood glucose.	**Interferences:** Hemolysis of sample, delayed separation of serum, prolonged fist clenching during blood drawing, and prolonged tourniquet placement. Very high WBC/PLT counts may cause spurious. Plasma potassium levels are normal.	**Interferences:**Test is helpful in assessing normal and increased anion gap metabolic acidosis and in distinguishing hypercalcemia due to hyperparathyroidism (high serum chloride) from that due to malignancy (Normal serum chloride)

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 g 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.

Dr. Akansha Jain
Consultant Pathologist

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

GLUCOSE, POST-PRANDIAL, PLASMA-High fasting glucose level in comparison to post prandial glucose level may beseen due to effect of Oral Hypoglycaemics & Insulin treatment, Renal Glycosuria, Glycaemic index & response to food consumed, Alimentary Hypoglycemia, Increased insulin response & sensitivity etc.Additional test HbA1c

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

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, Muscuophy

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,Multiple Sclerosis

TOTAL PROTEIN, SERUM-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, 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.

Dr. Akansha Jain
Consultant Pathologist

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CLINICAL PATH - URINALYSIS

MEDI WHEEL FULL BODY HEALTH CHECKUP ABOVE 40FEMALE

PHYSICAL EXAMINATION, URINE

COLOR METHOD : GROSS EXAMINATION	PALE YELLOW		
APPEARANCE METHOD : GROSS EXAMINATION	**SLIGHTLY HAZY**		

CHEMICAL EXAMINATION, URINE

PH METHOD : DOUBLE INDICATOR PRINCIPLE	5.5	4.7 - 7.5	
SPECIFIC GRAVITY METHOD : IONIC CONCENTRATION METHOD	<=1.005	1.003 - 1.035	
PROTEIN METHOD : PROTEIN ERROR OF INDICATORS WITH REFLECTANCE	NOT DETECTED	NEGATIVE	
GLUCOSE METHOD : GLUCOSE OXIDASE PEROXIDASE / BENEDICTS	NOT DETECTED	NEGATIVE	
KETONES METHOD : SODIUM NITROPRUSSIDE REACTION	NOT DETECTED	NOT DETECTED	
BLOOD METHOD : PEROCIDASE ANTI PEROXIDASE	NOT DETECTED	NOT DETECTED	
BILIRUBIN METHOD : DIPSTICK	NOT DETECTED	NOT DETECTED	
UROBILINOGEN METHOD : EHRLICH REACTION REFLECTANCE	NORMAL	NORMAL	
NITRITE METHOD : NITRATE TO NITRITE CONVERSION METHOD	NOT DETECTED	NOT DETECTED	
LEUKOCYTE ESTERASE	NOT DETECTED	NOT DETECTED	

MICROSCOPIC EXAMINATION, URINE

RED BLOOD CELLS METHOD : MICROSCOPIC EXAMINATION	NOT DETECTED	NOT DETECTED	/HPF
PUS CELL (WBC'S) METHOD : DIPSTICK, MICROSCOPY	2-3	0-5	/HPF

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EPITHELIAL CELLS	**5-7**	0-5	/HPF
METHOD : MICROSCOPIC EXAMINATION			
CASTS	NOT DETECTED		
METHOD : MICROSCOPIC EXAMINATION			
CRYSTALS	NOT DETECTED		
METHOD : MICROSCOPIC EXAMINATION			
BACTERIA	NOT DETECTED	NOT DETECTED	
METHOD : MICROSCOPIC EXAMINATION			
YEAST	NOT DETECTED	NOT DETECTED	

Interpretation(s)

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

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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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CYTOLOGY

MEDI WHEEL FULL BODY HEALTH CHECKUP ABOVE 40FEMALE

PAPANICOLAOU SMEAR

TEST METHOD	CONVENTIONAL GYNEC CYTOLOGY
SPECIMEN TYPE	TWO UNSTAINED CERVICAL SMEARS RECEIVED
REPORTING SYSTEM	2014 BETHESDA SYSTEM FOR REPORTING CERVICAL CYTOLOGY
SPECIMEN ADEQUACY	SMEARS ARE SATISFACTORY FOR EVALUATION.
MICROSCOPY	SMEARS SHOW PARABASAL, SUPERFICIAL AND INTERMEDIATE SQUAMOUS EPITHELIAL CELLS AGAINST MILD ACUTE INFLAMMATION .ENDO CERVICAL CELLS NOT SEEN . NO DYSPLASIA /MALIGNANCY SEEN.
METHOD : MICROSCOPY	
INTERPRETATION / RESULT	NEGATIVE FOR INTRAEPITHELIAL LESION OR MALIGNANCY
-	ATROPHY
METHOD : MANUAL	

Dr. Akansha Jain
Consultant Pathologist

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CLINICAL PATH - STOOL ANALYSIS

MEDI WHEEL FULL BODY HEALTH CHECKUP ABOVE 40FEMALE

PHYSICAL EXAMINATION,STOOL

COLOUR	SAMPLE NOT RECEIVED		

METHOD : GROSS EXAMINATION

Dr. Abhishek Sharma
Consultant Microbiologist

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SPECIALISED CHEMISTRY - HORMONE

MEDI WHEEL FULL BODY HEALTH CHECKUP ABOVE 40FEMALE

THYROID PANEL, SERUM

T3 METHOD : CHEMILUMINESCENCE	102.35	60.0 - 181.0	ng/dL
T4 METHOD : CHEMILUMINESCENCE	9.30	4.5 - 10.9	µg/dL
TSH (ULTRASENSITIVE) METHOD : CHEMILUMINESCENCE	2.230	0.550 - 4.780	µIU/mL

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 hyperthyroidism, 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, FreeT4 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
2	High	Normal	Normal	Normal	(1)Subclinical Hypothyroidism (2) Patient with insufficient thyroid 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

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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.

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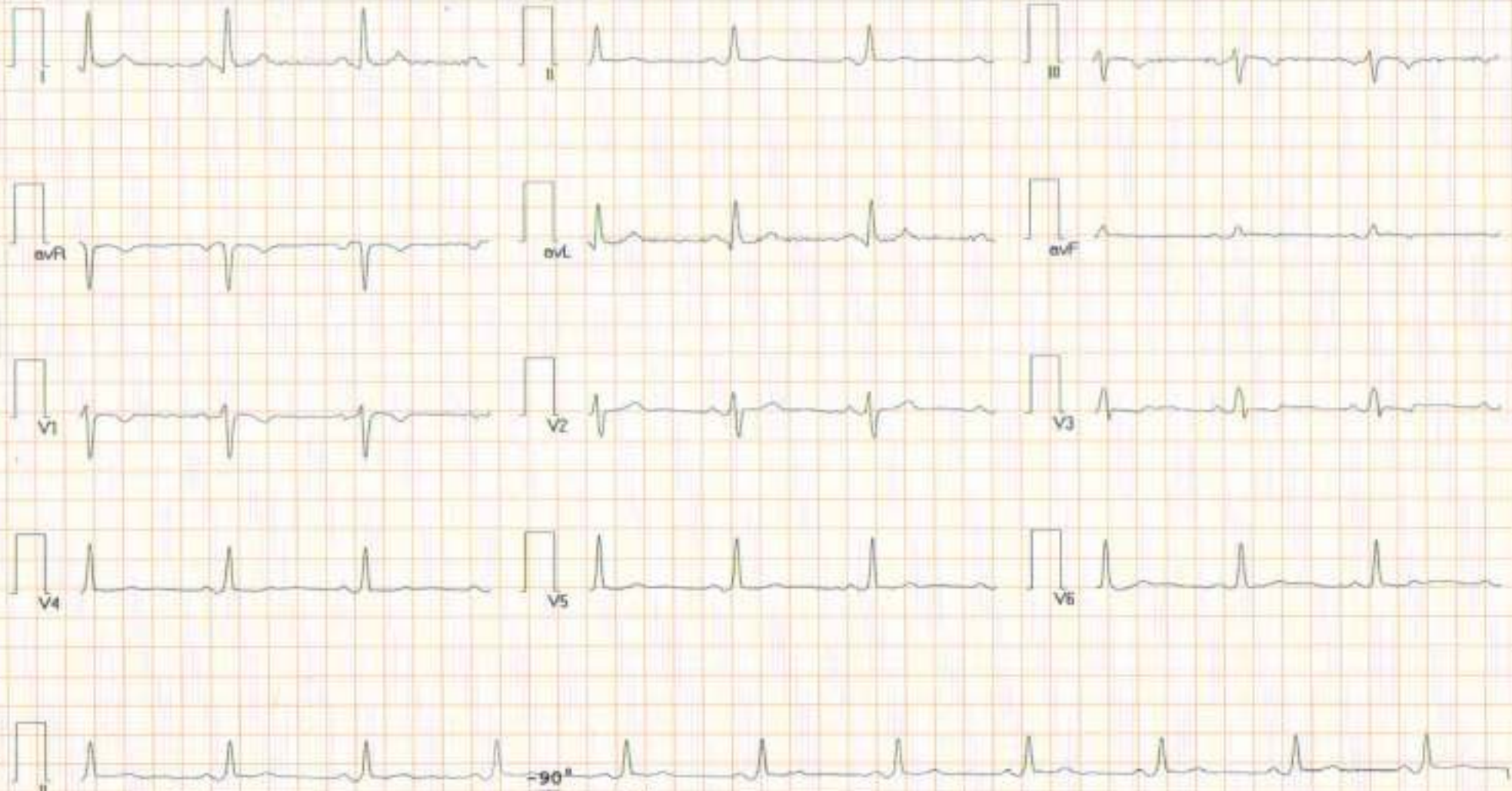
ECG

71467 / MRS NISHI GUPTA / 56 Yrs / F/ Non Smoker
Heart Rate : 65 bpm / Tested On : 27-Jan-24 09:37:15 / HF 0.05 Hz - LF 35 Hz / Notch 50 Hz / Sn 1.00 Cm/mV / Sw 25 mm/s
/ Refd By: MADI WHEEL

AGHPL



Vent Rate : 65 bpm
PR Interval : 144 ms
QRS Duration: 86 ms
QT/QTc Int : 378/387 ms
P-QRS-T axis: 12.00• 19.00• 6.00•

Axis
R 19.00° T 6.00° F 12.00°

Flattened ST segment

Dr. NITIZ GOYAL
M.B.B.S., M.D.
RMC - 023319

Reported By:DR NITIZ GOYAL

Allengers ECG (Pisces)(PIS218210312)

Aakriti Labs

wellness partner

3 Mahatma Gandhi Marg, Gandhi Nagar Mod
Tonk Road, Jaipur (Raj.) Ph.: 0141-2710661
www.aakritilabs.com
CIN NO.: U85195RJ2004PTC019563

NAME	MRS NISHI GUPTA	AGE	58Y	SEX	FEMALE
REF BY	MEDI WHEEL	DATE	27/01/2024	REG NO	

ECHOCARDIOGRAM REPORT

WINDOW- POOR/ADEQUATE/GOODVALVE

MITRAL	NORMAL	TRICUSPID	NORMAL
AORTIC	NORMAL	PULMONARY	NORMAL

2D/M-MOD

IVSD mm	9.5	IVSS mm	13.5	AORTA mm	23.0
LVID mm	35.9	LVIS mm	23.3	LA mm	29.4
LVPWD mm	9.8	LVPWS mm	14.2	EF%	60%

CHAMBERS

LA	NORMAL	RA	NORMAL
LV	NORMAL	RV	NORMAL
PERICARDIUM	NORMAL		

DOPPLER STUDY MITRAL

PEAK VELOCITY m/s E/A	0.93/0.78	PEAK GRADIANT MmHg	
MEAN VELOCITY m/s		MEAN GRADIANT MmHg	
MVA cm2 (PLANITMETERY)		MVA cm2 (PHT)	
MR			

AORTIC

PEAK VELOCITY m/s	1.18	PEAK GRADIANT MmHg	
MEAN VELOCITY m/s		MEAN GRADIANT MmHg	
AR			

TRICUSPID

PEAK VELOCITY m/s	1.02	PEAK GRADIANT MmHg	
MEAN VELOCITY m/s		MEAN GRADIANT MmHg	
TR	TRACE	PASP mmHg	

PULMONARY

PEAK VELOCITY m/s	0.90	PEAK GRADIANT MmHg	
MEAN VELOCITY m/s		MEAN GRADIANT MmHg	
PR		RVEDP mmHg	

IMPRESSION

- LV DAISTOLIC DYSFUNCTION GRADE-1
- NORMAL LV SYSTOLIC FUNCTION
- NO RWMA LVEF 60%
- NORMAL RV FUNCTION
- TRACE TR
- NORMAL CHAMBER DIMENSIONS
- NORMAL VALVULAR ECHO
- INTACT IAS / IVS
- NO THROMBUS, NO VEGETATION, NORMAL PERICARDIUM.
- IVC NORMAL

CONCLUSION : DAISTOLIC DYSFUNCTION,LV FUNCTION.

Cardiologist

Aakriti Labs

wellness partner

3 Mahatma Gandhi Marg, Gandhi Nagar Mod
Tonk Road, Jaipur (Raj.) Ph.: 0141-2710661
www.aakritilabs.com
CIN NO.: U85195RJ2004PTC019563

Name	: **Ms. NISHI GUPTA**	Registration No:	13216
Age/Gender:	56 Y 9 M 4 D/Female	Registered	: 27/Jan/2024 08:54AM
Patient ID	: 012401270013	Analysed	: 27/Jan/2024 11:10AM
BarcodeNo	:10112996	Reported	: 27/Jan/2024 11:10AM
Referred By	: Self	Panel	: ACROFEMI HEALTHCARE LTD (MEDIWHEEL)

OPHTHALMIC VISION TESTING

	RIGHT EYE	LEFT EYE
UCVA	6/36	6/24P
COLOURS	clear	clear
FUNDUS	WNL	WNL

	RIGHT EYE					LEFT EYE				
	SPH	CYL	AXIS	NEAR ADD	AV	SPH	CYL	AXIS	NEAR ADD	AV
PG	-1.00			6/6		-1.25				6/6
ACCEPTANCE	+0.25				N/6	+0.75				N/6
DILATED		←								
ADVISE										

Dr. RAKESH SHARMA
M.S. OPTH. & OPTH
FICLLP

*** End Of Report ***

wellness partner

Aakriti Labs

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Tonk Road, Jaipur (Raj.) Ph.: 0141-2710661
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CIN NO.: U85195RJ2004PTC019563

Name	: Ms. NISHI GUPTA	Registration No:	13216
Age/Gender:	56 Y 9 M 4 D/Female	Registered	: 27/Jan/2024 08:54AM
Patient ID	: 012401270013	Analysed	: 27/Jan/2024 10:25AM
BarcodeNo	: 10112996	Reported	: 27/Jan/2024 10:25AM
Referred By	: Self	Panel	: ACROFEMI HEALTHCARE LTD (MEDIWHEEL)

USG: WHOLE ABDOMEN (Female)

LIVER : Is normal in size, shape and echogenecity.
The IHBR and hepatic radicals are not dilated.
No evidence of focal echopoor/echorich lesion seen.
Portal vein diameter and Common bile duct normal in size

GALL BLADDER : Is not visualized. H/o Cholecystectomy.

PANCREAS: Is normal in size,shape and echotexture. Pancreatic duct is not dilated.

SPLEEN : Is normal in size,shape and echogenecity.Spleenic hilum is not dilated.

KIDNEYS : Right Kidney:-Size: 91 x 36 mm, Left Kidney:-Size: 114 x 42 mm.
Bilateral Kidneys are normal in size, shape and echotexture.
corticomedullary differentiation is fair and ratio appears normal.
Pelvi calyceal system is normal.No evidence of hydronephrosis/ nephrolithiasis.

URINARY BLADDER : Bladder walls are smooth,regular and normal thickness.
: No evidence of mass or stone in bladder lumen.

UTERUS : Uterus is anteverted with normal in size shape & echotexture.
Uterine muscular shadows normal echopattern.
Endometrium is normal and centrally placed.
No evidence of mass lesion is seen.

ADNEXA : Both the ovaries are normal in size shape and echotexture.
No mass lesion/ polycystic ovarian cyst is seen.

SPECIFIC : No evidence of retroperitoneal mass or free fluid seen in peritoneal cavity.
NO evidence of lymphadenopathy or mass lesion in retroperitoneum.
Visualized bowel loop appear normal.Great vessels appear normal.

IMPRESSION: Ultra Sonography findings are suggestive of: **NORMAL STUDY.**

Dr. Neera Mehta
M.B.B.S.,D.M.R.D.
RMCNO.005807/14853

Aakriti Labs

wellness partner

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CIN NO.: U85195RJ2004PTC019563

PATIENT NAME: MRS NISHI GUPTA	AGE: 56 Yrs.
REF. by: MEDIWHEEL	DATE: 27/01/2024

Ultrasonography report: Breast and Axilla

Findings:

Right Breast:-

Skin, subcutaneous tissue and retroareolar region is normal.

Fibroglandular tissue shows normal architecture and echotexture.

Pre and retromammary regions are unremarkable.

No obvious cyst, mass or architectural distortion visualized.

Axillary lymphnodes are not significantly enlarged and their hilar shadows are preserved.

Left Breast:-

Skin, subcutaneous tissue and retroareolar region is normal.

Fibroglandular tissue shows normal architecture and echotexture.

Pre and retromammary regions are unremarkable.

No obvious cyst, mass or architectural distortion visualized.

Axillary lymphnodes are not significantly enlarged and their hilar shadows are preserved.

IMPRESSION: No abnormality detected.

DR NEERA MEHTA
MBBS, DMRD
RMCNO.005807/14853

*****

PATIENT NAME : NISHI GUPTA | **REF. DOCTOR :** SELF

CODE/NAME & ADDRESS :C000049066
AGILUS DIAGNOSTICS LIMITED-WEL WALK-IN-
AAKRITI LABS PVT LTD. A-430, AGRASEN MARG
JAIPUR 302017
9314660100

ACCESSION NO : **0251XA002187**
PATIENT ID : NISHF231068251
CLIENT PATIENT ID: 012401270013
ABHA NO :

AGE/SEX : 56 Years Female
DRAWN : 27/01/2024 08:54:00
RECEIVED : 27/01/2024 09:31:19
REPORTED : 28/01/2024 09:46:28

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

HAEMATOLOGY - CBC

MEDI WHEEL FULL BODY HEALTH CHECKUP ABOVE 40FEMALE

BLOOD COUNTS,EDTA WHOLE BLOOD

HEMOGLOBIN (HB) METHOD : CYANIDE FREE DETERMINATION	14.0	12.0 - 15.0	g/dL
RED BLOOD CELL (RBC) COUNT METHOD : ELECTRICAL IMPEDANCE	**5.48 High**	3.8 - 4.8	mil/µL
WHITE BLOOD CELL (WBC) COUNT METHOD : ELECTRICAL IMPEDANCE	6.50	4.0 - 10.0	thou/µL
PLATELET COUNT METHOD : ELECTRONIC IMPEDANCE	189	150 - 410	thou/µL

RBC AND PLATELET INDICES

HEMATOCRIT (PCV) METHOD : CALCULATED PARAMETER	44.6	36 - 46	%
MEAN CORPUSCULAR VOLUME (MCV) METHOD : CALCULATED PARAMETER	**81.0 Low**	83 - 101	fL
MEAN CORPUSCULAR HEMOGLOBIN (MCH) METHOD : CALCULATED PARAMETER	**25.6 Low**	27.0 - 32.0	pg
MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION (MCHC) METHOD : CALCULATED PARAMETER	**31.4 Low**	31.5 - 34.5	g/dL
RED CELL DISTRIBUTION WIDTH (RDW) METHOD : CALCULATED PARAMETER	**16.3 High**	11.6 - 14.0	%
MENTZER INDEX	14.8		
MEAN PLATELET VOLUME (MPV) METHOD : CALCULATED PARAMETER	10.6	6.8 - 10.9	fL

WBC DIFFERENTIAL COUNT

NEUTROPHILS METHOD : IMPEDANCE WITH HYDRO FOCUS AND MICROSCOPY	58	40 - 80	%
LYMPHOCYTES METHOD : IMPEDANCE WITH HYDRO FOCUS AND MICROSCOPY	35	20 - 40	%
MONOCYTES	02	2 - 10	%

Dr. Akansha Jain
Consultant Pathologist

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Rajasthan, India

Patient Ref. No. 775000006205912

PATIENT NAME : NISHI GUPTA | **REF. DOCTOR :** SELF

CODE/NAME & ADDRESS :C000049066 AGILUS DIAGNOSTICS LIMITED-WEL WALK-IN-AAKRITI LABS PVT LTD. A-430, AGRASEN MARG JAIPUR 302017 9314660100	ACCESSION NO : **0251XA002187** PATIENT ID : NISHF231068251 CLIENT PATIENT ID: 012401270013 ABHA NO :	AGE/SEX : 56 Years Female DRAWN :27/01/2024 08:54:00 RECEIVED : 27/01/2024 09:31:19 REPORTED :28/01/2024 09:46:28

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METHOD : IMPEDANCE WITH HYDRO FOCUS AND MICROSCOPY EOSINOPHILS	05	1 - 6	%
METHOD : IMPEDANCE WITH HYDRO FOCUS AND MICROSCOPY BASOPHILS	00	0 - 2	%
METHOD : IMPEDANCE WITH HYDRO FOCUS AND MICROSCOPY ABSOLUTE NEUTROPHIL COUNT	3.77	2.0 - 7.0	thou/µL
METHOD : CALCULATED PARAMETER ABSOLUTE LYMPHOCYTE COUNT	2.28	1.0 - 3.0	thou/µL
METHOD : CALCULATED PARAMETER ABSOLUTE MONOCYTE COUNT	**0.13 Low**	0.2 - 1.0	thou/µL
METHOD : CALCULATED PARAMETER ABSOLUTE EOSINOPHIL COUNT	0.32	0.02 - 0.50	thou/µL
METHOD : CALCULATED PARAMETER ABSOLUTE BASOPHIL COUNT	**0 Low**	0.02 - 0.10	thou/µL
NEUTROPHIL LYMPHOCYTE RATIO (NLR)	1.7		

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.

Dr. Akansha Jain
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Patient Ref. No. 775000009205912

PATIENT NAME : NISHI GUPTA **REF. DOCTOR :** SELF

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HAEMATOLOGY

MEDI WHEEL FULL BODY HEALTH CHECKUP ABOVE 40FEMALE

GLYCOSYLATED HEMOGLOBIN(HBA1C), EDTA WHOLE BLOOD

HBA1C	5.6	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)	114.0	< 116.0	mg/dL
METHOD : CALCULATED PARAMETER			

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MEDI WHEEL FULL BODY HEALTH CHECKUP ABOVE 40FEMALE

ERYTHROCYTE SEDIMENTATION RATE (ESR),EDTA BLOOD

E.S.R	19	0 - 20	mm at 1 hr

METHOD : AUTOMATED (PHOTOMETRICAL CAPILLARY STOPPED FLOW KINETIC ANALYSIS)"

Interpretation(s)
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

ERYTHROCYTE SEDIMENTATION RATE (ESR),EDTA 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 ESR 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: Polycythemia 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.

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IMMUNOHAEMATOLOGY

MEDI WHEEL FULL BODY HEALTH CHECKUP ABOVE 40FEMALE

ABO GROUP & RH TYPE, EDTA WHOLE BLOOD

ABO GROUP METHOD : TUBE AGGLUTINATION	TYPE B		
RH TYPE METHOD : TUBE AGGLUTINATION	POSITIVE		

Interpretation(s)
ABO GROUP & RH TYPE, EDTA WHOLE BLOOD-Blood group is identified byantigens and antibodies present in the blood. Antigens are protein molecules found on the surface of red blood cells. Antibodies are found in plasma. To determine blood group, red cells are mixed with different antibody solutions to give A,B,O or AB.

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

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

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BIOCHEMISTRY

MEDI WHEEL FULL BODY HEALTH CHECKUP ABOVE 40FEMALE

GLUCOSE FASTING,FLUORIDE PLASMA

FBS (FASTING BLOOD SUGAR) METHOD : GLUCOSE OXIDASE	95	74 - 99	mg/dL

GLUCOSE, POST-PRANDIAL, PLASMA

PPBS(POST PRANDIAL BLOOD SUGAR) METHOD : GLUCOSE OXIDASE	115	70 - 140	mg/dL

LIPID PROFILE WITH CALCULATED LDL

CHOLESTEROL, TOTAL METHOD : CHOLESTEROL OXIDASE	183	< 200 Desirable 200 - 239 Borderline High >/= 240 High	mg/dL
TRIGLYCERIDES METHOD : LIPASE/GPO-PAP NO CORRECTION	84	< 150 Normal 150 - 199 Borderline High 200 - 499 High >/=500 Very High	mg/dL
HDL CHOLESTEROL METHOD : DIRECT CLEARANCE METHOD	42	< 40 Low >/=60 High	mg/dL
CHOLESTEROL LDL	**124 High**	< 100 Optimal 100 - 129 Near optimal/ above optimal 130 - 159 Borderline High 160 - 189 High >/= 190 Very High	mg/dL
NON HDL CHOLESTEROL METHOD : CALCULATED PARAMETER	**141 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	16.8	</= 30.0	mg/dL
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	
LDL/HDL RATIO	3.0	0.5 - 3.0 Desirable/Low Risk 3.1 - 6.0 Borderline/Moderate Risk >6.0 High Risk	

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 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 major risk factors or evidence of end organ damage 3. Familial Homozygous Hypercholesterolemia
High Risk	1. Three major ASCVD risk factors. 2. Diabetes 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	

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

Risk Group	**Treatment Goals**		**Consider Drug Therapy**	
	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)	>OR = 50	>OR = 80

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Extreme Risk Group Category B	<OR = 30	<OR = 60	> 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.

LIVER FUNCTION PROFILE, SERUM

BILIRUBIN, TOTAL	0.55	0 - 1	mg/dL
METHOD : DIAZO WITH SULPHANILIC ACID			
BILIRUBIN, DIRECT	0.19	0.00 - 0.25	mg/dL
METHOD : DIAZO WITH SULPHANILIC ACID			
BILIRUBIN, INDIRECT	0.36	0.1 - 1.0	mg/dL
METHOD : CALCULATED PARAMETER			
TOTAL PROTEIN	7.9	6.4 - 8.2	g/dL
METHOD : BIURET REACTION, END POINT			
ALBUMIN	**4.5 High**	3.8 - 4.4	g/dL
METHOD : BROMOCRESOL GREEN			
GLOBULIN	3.4	2.0 - 4.1	g/dL
METHOD : CALCULATED PARAMETER			
ALBUMIN/GLOBULIN RATIO	1.3	1.0 - 2.1	RATIO
METHOD : CALCULATED PARAMETER			
ASPARTATE AMINOTRANSFERASE(AST/SGOT)	**37 High**	0 - 31	U/L
METHOD : TRIS BUFFER NO P5P IFCC / SFBC 37° C			
ALANINE AMINOTRANSFERASE (ALT/SGPT)	**37 High**	0 - 31	U/L
METHOD : TRIS BUFFER NO P5P IFCC / SFBC 37° C			
ALKALINE PHOSPHATASE	96	39 - 117	U/L
METHOD : AMP OPTIMISED TO IFCC 37° C			
GAMMA GLUTAMYL TRANSFERASE (GGT)	23	7 - 32	U/L
METHOD : GAMMA GLUTAMYL-3 CARBOXY-4 NITROANILIDE (IFCC) 37° C			
LACTATE DEHYDROGENASE	343	230 - 460	U/L

BLOOD UREA NITROGEN (BUN), SERUM

BLOOD UREA NITROGEN	12	5.0 - 18.0	mg/dL
METHOD : UREASE KINETIC			

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Patient Ref. No. 775000006205912

PATIENT NAME : NISHI GUPTA **REF. DOCTOR :** SELF

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Test Report Status **Final**	Results	Biological Reference Interval	Units
CREATININE, SERUM			
CREATININE METHOD : ALKALINE PICRATE NO DEPROTEINIZATION	0.87	0.6 - 1.2	mg/dL
BUN/CREAT RATIO			
BUN/CREAT RATIO METHOD : CALCULATED PARAMETER	13.79		
URIC ACID, SERUM			
URIC ACID METHOD : URICASE PEROXIDASE WITH ASCORBATE OXIDASE	5.4	2.4 - 5.7	mg/dL
TOTAL PROTEIN, SERUM			
TOTAL PROTEIN METHOD : BIURET REACTION, END POINT	7.9	6.4 - 8.3	g/dL
ALBUMIN, SERUM			
ALBUMIN METHOD : BROMOCRESOL GREEN	**4.5 High**	3.8 - 4.4	g/dL
GLOBULIN			
GLOBULIN	3.4	2.0 - 4.1	g/dL
ELECTROLYTES (NA/K/CL), SERUM			

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SODIUM, SERUM METHOD : ION-SELECTIVE ELECTRODE	142.2	137 - 145	mmol/L
POTASSIUM, SERUM METHOD : ION-SELECTIVE ELECTRODE	4.30	3.6 - 5.0	mmol/L
CHLORIDE, SERUM METHOD : ION-SELECTIVE ELECTRODE	103.4	98 - 107	mmol/L

Interpretation(s)

Sodium	Potassium	Chloride
Decreased in:CCF,cirrhosis, vomiting, diarrhea, excessive sweating, salt-losing nephropathy,adrenal insufficiency, nephrotic syndrome, water intoxication, SIADH. Drugs: thiazides, diuretics, ACE inhibitors, chlorpropamide,carbamazepine,anti depressants (SSRI), antipsychotics.	**Decreased in:** Low potassium intake,prolonged vomiting or diarrhea, RTA types I and II, hyperaldosteronism, Cushing's syndrome,osmotic diuresis (e.g., hyperglycemia),alkalosis, familial periodic paralysis,trauma (transient).Drugs: Adrenergic agents, diuretics.	**Decreased in:** Vomiting, diarrhea, renal failure combined with salt deprivation, over-treatment with diuretics, chronic respiratory acidosis, diabetic ketoacidosis, excessive sweating, SIADH, salt-losing nephropathy, porphyria, expansion of extracellular fluid volume, adrenalinsufficiency, hyperaldosteronism,metabolic alkalosis. Drugs: chronic laxative,corticosteroids, diuretics.
Increased in: Dehydration (excessivesweating, severe vomiting or diarrhea),diabetes *mellitus, diabetesinsipidus,* hyperaldosteronism, inadequate water intake. Drugs: steroids, licorice,oral contraceptives.	**Increased in:** Massive hemolysis, severe tissue damage, rhabdomyolysis, acidosis, dehydration,renal failure, *Addison' s disease, RTA type IV,* hyperkalemic familial periodic paralysis. Drugs: potassium salts, potassium- sparing diuretics,NSAIDs, beta-blockers, ACE inhibitors, high-dose trimethoprim-sulfamethoxazole.	**Increased in:** Renal failure, nephrotic syndrome, RTA,dehydration, overtreatment with *saline,hyperparathyroidism, diabetes* insipidus, metabolic acidosis from diarrhea (Loss of HCO3-), respiratory alkalosis,hyperadrenocorticism. Drugs: acetazolamide,androgens, hydrochlorothiazide,salicylates.
Interferences: Severe lipemia or hyperproteinemi, if sodium analysis involves a dilution step can cause spurious results. The serum sodium falls about 1.6 mEq/L for each 100 mg/dL increase in blood glucose.	**Interferences:** Hemolysis of sample, delayed separation of serum, prolonged fist clenching during blood drawing, and prolonged tourniquet placement. Very high WBC/PLT counts may cause spurious. Plasma potassium levels are normal.	**Interferences:**Test is helpful in assessing normal and increased anion gap metabolic acidosis and in distinguishing hypercalcemia due to hyperparathyroidism (high serum chloride) from that due to malignancy (Normal serum chloride)

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 g 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.

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

GLUCOSE, POST-PRANDIAL, PLASMA-High fasting glucose level in comparison to post prandial glucose level may beseen due to effect of Oral Hypoglycaemics & Insulin treatment, Renal Glycosuria, Glycaemic index & response to food consumed, Alimentary Hypoglycemia, Increased insulin response & sensitivity etc.Additional test HbA1c

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

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, Muscuophy

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,Multiple Sclerosis

TOTAL PROTEIN, SERUM-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, 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.

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CLINICAL PATH - URINALYSIS

MEDI WHEEL FULL BODY HEALTH CHECKUP ABOVE 40FEMALE

PHYSICAL EXAMINATION, URINE

COLOR METHOD : GROSS EXAMINATION	PALE YELLOW		
APPEARANCE METHOD : GROSS EXAMINATION	**SLIGHTLY HAZY**		

CHEMICAL EXAMINATION, URINE

PH METHOD : DOUBLE INDICATOR PRINCIPLE	5.5	4.7 - 7.5	
SPECIFIC GRAVITY METHOD : IONIC CONCENTRATION METHOD	<=1.005	1.003 - 1.035	
PROTEIN METHOD : PROTEIN ERROR OF INDICATORS WITH REFLECTANCE	NOT DETECTED	NEGATIVE	
GLUCOSE METHOD : GLUCOSE OXIDASE PEROXIDASE / BENEDICTS	NOT DETECTED	NEGATIVE	
KETONES METHOD : SODIUM NITROPRUSSIDE REACTION	NOT DETECTED	NOT DETECTED	
BLOOD METHOD : PEROCIDASE ANTI PEROXIDASE	NOT DETECTED	NOT DETECTED	
BILIRUBIN METHOD : DIPSTICK	NOT DETECTED	NOT DETECTED	
UROBILINOGEN METHOD : EHRLICH REACTION REFLECTANCE	NORMAL	NORMAL	
NITRITE METHOD : NITRATE TO NITRITE CONVERSION METHOD	NOT DETECTED	NOT DETECTED	
LEUKOCYTE ESTERASE	NOT DETECTED	NOT DETECTED	

MICROSCOPIC EXAMINATION, URINE

RED BLOOD CELLS METHOD : MICROSCOPIC EXAMINATION	NOT DETECTED	NOT DETECTED	/HPF
PUS CELL (WBC'S) METHOD : DIPSTICK, MICROSCOPY	2-3	0-5	/HPF

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EPITHELIAL CELLS	**5-7**	0-5	/HPF
METHOD : MICROSCOPIC EXAMINATION			
CASTS	NOT DETECTED		
METHOD : MICROSCOPIC EXAMINATION			
CRYSTALS	NOT DETECTED		
METHOD : MICROSCOPIC EXAMINATION			
BACTERIA	NOT DETECTED	NOT DETECTED	
METHOD : MICROSCOPIC EXAMINATION			
YEAST	NOT DETECTED	NOT DETECTED	

Interpretation(s)

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

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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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CYTOLOGY

MEDI WHEEL FULL BODY HEALTH CHECKUP ABOVE 40FEMALE

PAPANICOLAOU SMEAR

TEST METHOD	CONVENTIONAL GYNEC CYTOLOGY
SPECIMEN TYPE	TWO UNSTAINED CERVICAL SMEARS RECEIVED
REPORTING SYSTEM	2014 BETHESDA SYSTEM FOR REPORTING CERVICAL CYTOLOGY
SPECIMEN ADEQUACY	SMEARS ARE SATISFACTORY FOR EVALUATION.
MICROSCOPY	SMEARS SHOW PARABASAL, SUPERFICIAL AND INTERMEDIATE SQUAMOUS EPITHELIAL CELLS AGAINST MILD ACUTE INFLAMMATION .ENDO CERVICAL CELLS NOT SEEN . NO DYSPLASIA /MALIGNANCY SEEN.
METHOD : MICROSCOPY	
INTERPRETATION / RESULT	NEGATIVE FOR INTRAEPITHELIAL LESION OR MALIGNANCY
-	ATROPHY
METHOD : MANUAL	

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CLINICAL PATH - STOOL ANALYSIS

MEDI WHEEL FULL BODY HEALTH CHECKUP ABOVE 40FEMALE

PHYSICAL EXAMINATION,STOOL

COLOUR	SAMPLE NOT RECEIVED		

METHOD : GROSS EXAMINATION

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SPECIALISED CHEMISTRY - HORMONE

MEDI WHEEL FULL BODY HEALTH CHECKUP ABOVE 40FEMALE

THYROID PANEL, SERUM

T3 METHOD : CHEMILUMINESCENCE	102.35	60.0 - 181.0	ng/dL
T4 METHOD : CHEMILUMINESCENCE	9.30	4.5 - 10.9	µg/dL
TSH (ULTRASENSITIVE) METHOD : CHEMILUMINESCENCE	2.230	0.550 - 4.780	µIU/mL

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 hyperthyroidism, 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, FreeT4 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
2	High	Normal	Normal	Normal	(1)Subclinical Hypothyroidism (2) Patient with insufficient thyroid 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

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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

Dr. Akansha Jain
Consultant Pathologist

View Details View Report

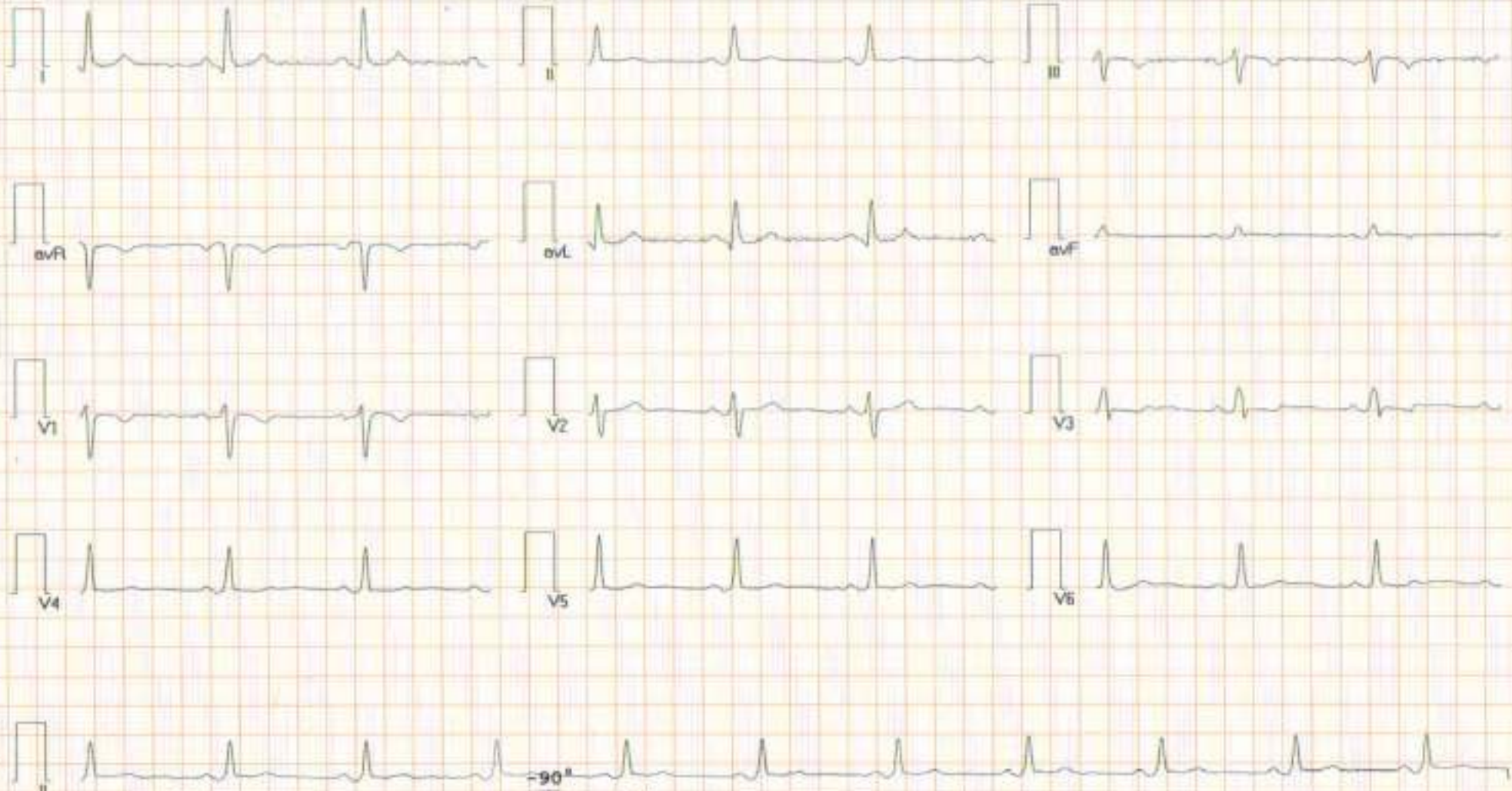
PERFORMED AT :
Agilus Diagnostics Ltd.
C/O Aakriti Labs Pvt Ltd, 3, Mahatma Gandhi Marg,Gandhi Nagar Mod, Tonk Road
Jaipur, 302015
Rajasthan, India

Patient Ref. No. 775000009205912

ECG

71467 / MRS NISHI GUPTA / 56 Yrs / F/ Non Smoker
Heart Rate : 65 bpm / Tested On : 27-Jan-24 09:37:15 / HF 0.05 Hz - LF 35 Hz / Notch 50 Hz / Sn 1.00 Cm/mV / Sw 25 mm/s
/ Refd By: MADI WHEEL

AGHPL



Vent Rate : 65 bpm
PR Interval : 144 ms
QRS Duration: 86 ms
QT/QTc Int : 378/387 ms
P-QRS-T axis: 12.00• 19.00• 6.00•

Axis
R 19.00° T 6.00° F 12.00°

Flattened ST segment

Dr. NITIZ GOYAL
M.B.B.S., M.D.
RMC - 023319

Reported By:DR NITIZ GOYAL

Allengers ECG (Pisces)(PIS218210312)

Aakriti Labs

wellness partner

3 Mahatma Gandhi Marg, Gandhi Nagar Mod
Tonk Road, Jaipur (Raj.) Ph.: 0141-2710661
www.aakritilabs.com
CIN NO.: U85195RJ2004PTC019563

NAME	MRS NISHI GUPTA	AGE	58Y	SEX	FEMALE
REF BY	MEDI WHEEL	DATE	27/01/2024	REG NO	

ECHOCARDIOGRAM REPORT

WINDOW- POOR/ADEQUATE/GOODVALVE

MITRAL	NORMAL	TRICUSPID	NORMAL
AORTIC	NORMAL	PULMONARY	NORMAL

2D/M-MOD

IVSD mm	9.5	IVSS mm	13.5	AORTA mm	23.0
LVID mm	35.9	LVIS mm	23.3	LA mm	29.4
LVPWD mm	9.8	LVPWS mm	14.2	EF%	60%

CHAMBERS

LA	NORMAL	RA	NORMAL
LV	NORMAL	RV	NORMAL
PERICARDIUM	NORMAL		

DOPPLER STUDY MITRAL

PEAK VELOCITY m/s E/A	0.93/0.78	PEAK GRADIANT MmHg	
MEAN VELOCITY m/s		MEAN GRADIANT MmHg	
MVA cm2 (PLANITMETERY)		MVA cm2 (PHT)	
MR			

AORTIC

PEAK VELOCITY m/s	1.18	PEAK GRADIANT MmHg	
MEAN VELOCITY m/s		MEAN GRADIANT MmHg	
AR			

TRICUSPID

PEAK VELOCITY m/s	1.02	PEAK GRADIANT MmHg	
MEAN VELOCITY m/s		MEAN GRADIANT MmHg	
TR	TRACE	PASP mmHg	

PULMONARY

PEAK VELOCITY m/s	0.90	PEAK GRADIANT MmHg	
MEAN VELOCITY m/s		MEAN GRADIANT MmHg	
PR		RVEDP mmHg	

IMPRESSION

- LV DAISTOLIC DYSFUNCTION GRADE-1
- NORMAL LV SYSTOLIC FUNCTION
- NO RWMA LVEF 60%
- NORMAL RV FUNCTION
- TRACE TR
- NORMAL CHAMBER DIMENSIONS
- NORMAL VALVULAR ECHO
- INTACT IAS / IVS
- NO THROMBUS, NO VEGETATION, NORMAL PERICARDIUM.
- IVC NORMAL

CONCLUSION : DAISTOLIC DYSFUNCTION,LV FUNCTION.

Cardiologist

Aakriti Labs

wellness partner

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www.aakritilabs.com
CIN NO.: U85195RJ2004PTC019563

Name	: **Ms. NISHI GUPTA**	Registration No:	13216
Age/Gender:	56 Y 9 M 4 D/Female	Registered	: 27/Jan/2024 08:54AM
Patient ID	: 012401270013	Analysed	: 27/Jan/2024 11:10AM
BarcodeNo	:10112996	Reported	: 27/Jan/2024 11:10AM
Referred By	: Self	Panel	: ACROFEMI HEALTHCARE LTD (MEDIWHEEL)

OPHTHALMIC VISION TESTING

	RIGHT EYE	LEFT EYE
UCVA	6/36	6/24P
COLOURS	clear	clear
FUNDUS	WNL	WNL

	RIGHT EYE					LEFT EYE				
	SPH	CYL	AXIS	NEAR ADD	AV	SPH	CYL	AXIS	NEAR ADD	AV
PG	-1.00			6/6		-1.25				6/6
ACCEPTANCE	+0.25				N/6	+0.75				N/6
DILATED		←								
ADVISE										

Dr. RAKESH SHARMA
M.S. OPTH. & OPTH
FICLLP

*** End Of Report ***

wellness partner

Aakriti Labs

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CIN NO.: U85195RJ2004PTC019563

Name	: Ms. NISHI GUPTA	Registration No:	13216
Age/Gender:	56 Y 9 M 4 D/Female	Registered	: 27/Jan/2024 08:54AM
Patient ID	: 012401270013	Analysed	: 27/Jan/2024 10:25AM
BarcodeNo	: 10112996	Reported	: 27/Jan/2024 10:25AM
Referred By	: Self	Panel	: ACROFEMI HEALTHCARE LTD (MEDIWHEEL)

USG: WHOLE ABDOMEN (Female)

LIVER : Is normal in size, shape and echogenecity.
The IHBR and hepatic radicals are not dilated.
No evidence of focal echopoor/echorich lesion seen.
Portal vein diameter and Common bile duct normal in size

GALL BLADDER : Is not visualized. H/o Cholecystectomy.

PANCREAS: Is normal in size,shape and echotexture. Pancreatic duct is not dilated.

SPLEEN : Is normal in size,shape and echogenecity.Spleenic hilum is not dilated.

KIDNEYS : Right Kidney:-Size: 91 x 36 mm, Left Kidney:-Size: 114 x 42 mm.
Bilateral Kidneys are normal in size, shape and echotexture.
corticomedullary differentiation is fair and ratio appears normal.
Pelvi calyceal system is normal.No evidence of hydronephrosis/ nephrolithiasis.

URINARY BLADDER : Bladder walls are smooth,regular and normal thickness.
: No evidence of mass or stone in bladder lumen.

UTERUS : Uterus is anteverted with normal in size shape & echotexture.
Uterine muscular shadows normal echopattern.
Endometrium is normal and centrally placed.
No evidence of mass lesion is seen.

ADNEXA : Both the ovaries are normal in size shape and echotexture.
No mass lesion/ polycystic ovarian cyst is seen.

SPECIFIC : No evidence of retroperitoneal mass or free fluid seen in peritoneal cavity.
NO evidence of lymphadenopathy or mass lesion in retroperitoneum.
Visualized bowel loop appear normal.Great vessels appear normal.

IMPRESSION: Ultra Sonography findings are suggestive of: **NORMAL STUDY.**

Dr. Neera Mehta
M.B.B.S.,D.M.R.D.
RMCNO.005807/14853

Aakriti Labs

wellness partner

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Tonk Road, Jaipur (Raj.) Ph.: 0141-2710661
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CIN NO.: U85195RJ2004PTC019563

PATIENT NAME: MRS NISHI GUPTA	AGE: 56 Yrs.
REF. by: MEDIWHEEL	DATE: 27/01/2024

Ultrasonography report: Breast and Axilla

Findings:

Right Breast:-

Skin, subcutaneous tissue and retroareolar region is normal.

Fibroglandular tissue shows normal architecture and echotexture.

Pre and retromammary regions are unremarkable.

No obvious cyst, mass or architectural distortion visualized.

Axillary lymphnodes are not significantly enlarged and their hilar shadows are preserved.

Left Breast:-

Skin, subcutaneous tissue and retroareolar region is normal.

Fibroglandular tissue shows normal architecture and echotexture.

Pre and retromammary regions are unremarkable.

No obvious cyst, mass or architectural distortion visualized.

Axillary lymphnodes are not significantly enlarged and their hilar shadows are preserved.

IMPRESSION: No abnormality detected.

DR NEERA MEHTA
MBBS, DMRD
RMCNO.005807/14853

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