MC-5837

PATIENT NAME : MR.ABHINAV CHATTERJI	**REF. DOCTOR :**	
CODE/NAME & ADDRESS : C000045507 FORTIS VASHI-CHC -SPLZD FORTIS HOSPITAL # VASHI, MUMBAI 440001	ACCESSION NO : **0022WI006104** PATIENT ID : FH.12738337 CLIENT PATIENT ID: UID:12738337 ABHA NO :	AGE/SEX : 49 Years Male DRAWN : 29/09/2023 11:06:00 RECEIVED : 29/09/2023 11:06:38 REPORTED : 29/09/2023 14:29:31

CLINICAL INFORMATION :

UID:12738337 REQNO-1588048
CORP-OPD
BILLNO-150123OPCR055743
BILLNO-150123OPCR055743

Test Report Status **Final**	Results	Biological Reference Interval	Units
BIOCHEMISTRY			
GLUCOSE, POST-PRANDIAL, PLASMA			
PPBS(POST PRANDIAL BLOOD SUGAR)	96	70 - 140	mg/dL
METHOD : HEXOKINASE			

Comments

NOTE:- RECHECKED FOR POST PRANDIAL PLASMA GLUCOSE VALUES. TO BE CORRELATE WITH CLINICAL, DIETETIC AND THERAPEUTIC HISTORY.

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

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

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Consultant Pathologist

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Patient Ref. No. 220000000875371

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CODE/NAME & ADDRESS : C000045507 FORTIS VASHI-CHC -SPLZD FORTIS HOSPITAL # VASHI, MUMBAI 440001	ACCESSION NO : **0022WI006076** PATIENT ID : FH.12738337 CLIENT PATIENT ID: UID:12738337 ABHA NO :	AGE/SEX : 49 Years Male DRAWN : 29/09/2023 08:31:00 RECEIVED : 29/09/2023 08:31:27 REPORTED : 29/09/2023 14:00:33

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

HAEMATOLOGY - CBC

CBC-5, EDTA WHOLE BLOOD

BLOOD COUNTS, EDTA WHOLE BLOOD

HEMOGLOBIN (HB) METHOD : SLS METHOD	15.4	13.0 - 17.0	g/dL
RED BLOOD CELL (RBC) COUNT METHOD : HYDRODYNAMIC FOCUSING	5.03	4.5 - 5.5	mil/µL
WHITE BLOOD CELL (WBC) COUNT METHOD : FLUORESCENCE FLOW CYTOMETRY	4.11	4.0 - 10.0	thou/µL
PLATELET COUNT METHOD : HYDRODYNAMIC FOCUSING BY DC DETECTION	183	150 - 410	thou/µL

RBC AND PLATELET INDICES

HEMATOCRIT (PCV) METHOD : CUMULATIVE PULSE HEIGHT DETECTION METHOD	45.6	40.0 - 50.0	%
MEAN CORPUSCULAR VOLUME (MCV) METHOD : CALCULATED PARAMETER	90.7	83.0 - 101.0	fL
MEAN CORPUSCULAR HEMOGLOBIN (MCH) METHOD : CALCULATED PARAMETER	30.6	27.0 - 32.0	pg
MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION(MCHC) METHOD : CALCULATED PARAMETER	33.8	31.5 - 34.5	g/dL
RED CELL DISTRIBUTION WIDTH (RDW) METHOD : CALCULATED PARAMETER	13.0	11.6 - 14.0	%
MENTZER INDEX METHOD : CALCULATED PARAMETER	18.0		
MEAN PLATELET VOLUME (MPV) METHOD : CALCULATED PARAMETER	**11.7 High**	6.8 - 10.9	fL

WBC DIFFERENTIAL COUNT

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NEUTROPHILS METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING	50	40.0 - 80.0	%
LYMPHOCYTES METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING	39	20.0 - 40.0	%
MONOCYTES METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING	6	2.0 - 10.0	%
EOSINOPHILS METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING	5	1 - 6	%
BASOPHILS METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING	0	0 - 2	%
ABSOLUTE NEUTROPHIL COUNT METHOD : CALCULATED PARAMETER	2.06	2.0 - 7.0	thou/µL
ABSOLUTE LYMPHOCYTE COUNT METHOD : CALCULATED PARAMETER	1.60	1.0 - 3.0	thou/µL
ABSOLUTE MONOCYTE COUNT METHOD : CALCULATED PARAMETER	0.25	0.2 - 1.0	thou/µL
ABSOLUTE EOSINOPHIL COUNT METHOD : CALCULATED PARAMETER	0.21	0.02 - 0.50	thou/µL
ABSOLUTE BASOPHIL COUNT METHOD : CALCULATED PARAMETER	**0 Low**	0.02 - 0.10	thou/µL
NEUTROPHIL LYMPHOCYTE RATIO (NLR) METHOD : CALCULATED	1.3		

MORPHOLOGY

RBC METHOD : MICROSCOPIC EXAMINATION	PREDOMINANTLY NORMOCYTIC NORMOCHROMIC
WBC METHOD : MICROSCOPIC EXAMINATION	NORMAL MORPHOLOGY
PLATELETS METHOD : MICROSCOPIC EXAMINATION	ADEQUATE

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Interpretation(s)
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

ERYTHROCYTE SEDIMENTATION RATE (ESR),WHOLE BLOOD

E.S.R	04	0 - 14	mm at 1 hr

METHOD : WESTERGREN METHOD

GLYCOSYLATED HEMOGLOBIN(HBA1C), EDTA WHOLE BLOOD

HBA1C	5.5	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 : HB VARIANT (HPLC)

ESTIMATED AVERAGE GLUCOSE(EAG)	111.2	< 116.0	mg/dL

METHOD : CALCULATED PARAMETER

Interpretation(s)
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 BRI in first trimester is 0-48 mm/hr(62 if anemic) and in second trimester (0-70 mm /hr(95 if anemic). ESR returns to normal 4th week post partum.
Decreased in: Polycythermia vera, Sickle cell anemia

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

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REFERENCE :
1. Nathan and Oski's Haematology of Infancy and Childhood, 5th edition;2. Paediatric reference intervals. AACC Press, 7th edition. Edited by S. Soldin;3. The reference for the adult reference range is "Practical Haematology by Dacie and Lewis,10th edition.
GLYCOSYLATED HEMOGLOBIN(HBA1C), EDTA WHOLE BLOOD-**Used For**:

1. Evaluating the long-term control of blood glucose concentrations in diabetic patients.
2. Diagnosing diabetes.
3. Identifying patients at increased risk for diabetes (prediabetes).
The ADA recommends measurement of HbA1c (typically 3-4 times per year for type 1 and poorly controlled type 2 diabetic patients, and 2 times per year for well-controlled type 2 diabetic patients) to determine whether a patients metabolic control has remained continuously within the target range.
1. eAG (Estimated average glucose) converts percentage HbA1c to md/dl, to compare blood glucose levels.
2. eAG gives an evaluation of blood glucose levels for the last couple of months.
3. eAG is calculated as eAG (mg/dl) = 28.7 * HbA1c - 46.7

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

a) Homozygous hemoglobinopathy. Fructosamine is recommended for testing of HbA1c.
b) Heterozygous state detected (D10 is corrected for HbS & HbC trait.)
c) HbF > 25% on alternate paltform (Boronate affinity chromatography) is recommended for testing of HbA1c.Abnormal Hemoglobin electrophoresis (HPLC method) is recommended for detecting a hemoglobinopathy

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IMMUNOHAEMATOLOGY

ABO GROUP & RH TYPE, EDTA WHOLE BLOOD

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

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

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

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

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BIOCHEMISTRY			
LIVER FUNCTION PROFILE, SERUM			
BILIRUBIN, TOTAL	**1.18 High**	0.2 - 1.0	mg/dL
METHOD : JENDRASSIK AND GROFF			
BILIRUBIN, DIRECT	0.17	0.0 - 0.2	mg/dL
METHOD : JENDRASSIK AND GROFF			
BILIRUBIN, INDIRECT	**1.01 High**	0.1 - 1.0	mg/dL
METHOD : CALCULATED PARAMETER			
TOTAL PROTEIN	7.2	6.4 - 8.2	g/dL
METHOD : BIURET			
ALBUMIN	4.2	3.4 - 5.0	g/dL
METHOD : BCP DYE BINDING			
GLOBULIN	3.0	2.0 - 4.1	g/dL
METHOD : CALCULATED PARAMETER			
ALBUMIN/GLOBULIN RATIO	1.4	1.0 - 2.1	RATIO
METHOD : CALCULATED PARAMETER			
ASPARTATE AMINOTRANSFERASE(AST/SGOT)	20	15 - 37	U/L
METHOD : UV WITH P5P			
ALANINE AMINOTRANSFERASE (ALT/SGPT)	30	< 45.0	U/L
METHOD : UV WITH P5P			
ALKALINE PHOSPHATASE	77	30 - 120	U/L
METHOD : PNPP-ANP			
GAMMA GLUTAMYL TRANSFERASE (GGT)	24	15 - 85	U/L
METHOD : GAMMA GLUTAMYLCARBOXY 4NITROANILIDE			
LACTATE DEHYDROGENASE	152	85 - 227	U/L
METHOD : LACTATE -PYRUVATE			
GLUCOSE FASTING,FLUORIDE PLASMA			
FBS (FASTING BLOOD SUGAR)	**106 High**	Normal : < 100 Pre-diabetes: 100-125 Diabetes: >/=126	mg/dL
METHOD : HEXOKINASE			

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KIDNEY PANEL - 1			
BLOOD UREA NITROGEN (BUN), SERUM			
BLOOD UREA NITROGEN	13	6 - 20	mg/dL
METHOD : UREASE - UV			
CREATININE EGFR- EPI			
CREATININE	**1.31 High**	0.90 - 1.30	mg/dL
METHOD : ALKALINE PICRATE KINETIC JAFFES			
AGE	49		years
GLOMERULAR FILTRATION RATE (MALE)	66.73	Refer Interpretation Below	mL/min/1.73m2
METHOD : CALCULATED PARAMETER			
BUN/CREAT RATIO			
BUN/CREAT RATIO	9.92	5.00 - 15.00	
METHOD : CALCULATED PARAMETER			
URIC ACID, SERUM			
URIC ACID	6.1	3.5 - 7.2	mg/dL
METHOD : URICASE UV			
TOTAL PROTEIN, SERUM			
TOTAL PROTEIN	7.2	6.4 - 8.2	g/dL
METHOD : BIURET			

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ALBUMIN, SERUM			
ALBUMIN METHOD : BCP DYE BINDING	4.2	3.4 - 5.0	g/dL
GLOBULIN			
GLOBULIN METHOD : CALCULATED PARAMETER	3.0	2.0 - 4.1	g/dL
ELECTROLYTES (NA/K/CL), SERUM			
SODIUM, SERUM METHOD : ISE INDIRECT	136	136 - 145	mmol/L
POTASSIUM, SERUM METHOD : ISE INDIRECT	5.01	3.50 - 5.10	mmol/L
CHLORIDE, SERUM METHOD : ISE INDIRECT	100	98 - 107	mmol/L

Interpretation(s)

Interpretation(s)
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.

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AST is an enzyme found in various parts of the body. AST is found in the liver, heart, skeletal muscle, kidneys, brain, and red blood cells, and it is commonly measured clinically as a marker for liver health. AST levels increase during chronic viral hepatitis, blockage of the bile duct, cirrhosis of the liver,liver cancer,kidney failure,hemolytic anemia,pancreatitis,hemochromatosis. AST levels may also increase after a heart attack or strenuous activity.ALT test measures the amount of this enzyme in the blood.ALT is found mainly in the liver, but also in smaller amounts in the kidneys,heart,muscles, and pancreas.It is commonly measured as a part of a diagnostic evaluation of hepatocellular injury, to determine liver health.AST levels increase during acute hepatitis,sometimes due to a viral infection,ischemia to the liver,chronic hepatitis,obstruction of bile ducts,cirrhosis.
ALP is a protein found in almost all body tissues.Tissues with higher amounts of ALP include the liver,bile ducts and bone.Elevated ALP levels are seen in Biliary obstruction, Osteoblastic bone tumors, osteomalacia, hepatitis, Hyperparathyroidism, Leukemia, Lymphoma, 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
GLUCOSE FASTING,FLUORIDE PLASMA-**TEST DESCRIPTION**
Normally, the glucose concentration in extracellular fluid is closely regulated so that a source of energy is readily available to tissues and sothat no glucose is excreted in the urine.
Increased in:Diabetes mellitus, Cushing' s syndrome (10 – 15%), chronic pancreatitis (30%). Drugs:corticosteroids,phenytoin, estrogen, thiazides.
Decreased in :Pancreatic islet cell disease with increased insulin,insulinoma,adrenocortical insufficiency,hypopituitarism,diffuse liver disease, malignancy(adrenocortical,stomach,fibrosarcoma),infant of a diabetic mother,enzyme deficiency diseases(e.g.galactosemia),Drugs-insulin,ethanol,propranolol;sulfonylureas,tolbutamide,and other oral hypoglycemic agents.
NOTE: While random serum glucose levels correlate with home glucose monitoring results (weekly mean capillary glucose values),there is wide fluctuation within individuals.Thus, glycosylated hemoglobin(HbA1c) levels are favored to monitor glycemic control.
High fasting glucose level in comparison to post prandial glucose level may be seen due to effect of Oral Hypoglycaemics & Insulin treatment,Renal Glyosuria,Glycaemic index & response to food consumed,Alimentary Hypoglycemia,Increased insulin response & sensitivity etc.
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 EGFR- EPI-- Kidney disease outcomes quality initiative (KDOQI) guidelines state that estimation of GFR is the best overall indices of the Kidney function.
- It gives a rough measure of number of functioning nephrons .Reduction in GFR implies progression of underlying disease.
- The GFR is a calculation based on serum creatinine test.
- Creatinine is mainly derived from the metabolism of creatine in muscle, and its generation is proportional to the total muscle mass. As a result, mean creatinine generation is higher in men than in women, in younger than in older individuals, and in blacks than in whites.
- Creatinine is filtered from the blood by the kidneys and excreted into urine at a relatively steady rate.
- When kidney function is compromised, excretion of creatinine decreases with a consequent increase in blood creatinine levels. With the creatinine test, a reasonable estimate of the actual GFR can be determined.
- This equation takes into account several factors that impact creatinine production, including age, gender, and race.
- CKD EPI (Chronic kidney disease epidemiology collaboration) equation performed better than MDRD equation especially when GFR is high(>60 ml/min per 1.73m2).. This formula has less bias and greater accuracy which helps in early diagnosis and also reduces the rate of false positive diagnosis of CKD.

References:

National Kidney Foundation (NKF) and the American Society of Nephrology (ASN).
Estimated GFR Calculated Using the CKD-EPI equation-https://testguide.labmed.uw.edu/guideline/egfr
Ghuman JK, et al. Impact of Removing Race Variable on CKD Classification Using the Creatinine-Based 2021 CKD-EPI Equation. Kidney Med 2022, 4:100471. 35756325
Harrison''s Principle of Internal Medicine, 21st ed. pg 62 and 334
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.

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Email : -

Patient Ref. No. 22000000875343

PATIENT NAME : MR.ABHINAV CHATTERJI	**REF. DOCTOR :**	
CODE/NAME & ADDRESS : C000045507 FORTIS VASHI-CHC -SPLZD FORTIS HOSPITAL # VASHI, MUMBAI 440001	ACCESSION NO : **0022WI006076** PATIENT ID : FH.12738337 CLIENT PATIENT ID: UID:12738337 ABHA NO :	AGE/SEX : 49 Years Male DRAWN : 29/09/2023 08:31:00 RECEIVED : 29/09/2023 08:31:27 REPORTED : 29/09/2023 14:00:33

CLINICAL INFORMATION :

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Lower-than-normal levels may be due to: Agammaglobulinemia, Bleeding (hemorrhage),Burns,Glomerulonephritis, Liver disease, Malabsorption, Malnutrition, Nephrotic syndrome,Protein-losing enteropathy etc.
ALBUMIN, SERUM-Human serum albumin is the most abundant protein in human blood plasma. It is produced in the liver. Albumin constitutes about half of the blood serum protein. **Low blood albumin levels (hypoalbuminemia) can be caused by:** Liver disease like cirrhosis of the liver, nephrotic syndrome, protein-losing enteropathy, Burns, hemodilution, increased vascular permeability or decreased lymphatic clearance,malnutrition and wasting etc.

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MC-5837

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BIOCHEMISTRY - LIPID

LIPID PROFILE, SERUM

CHOLESTEROL, TOTAL	**242 High**	< 200 Desirable 200 - 239 Borderline High >/= 240 High	mg/dL
METHOD : ENZYMATIC/COLORIMETRIC,CHOLESTEROL OXIDASE, ESTERASE, PEROXIDASE			
TRIGLYCERIDES	122	< 150 Normal 150 - 199 Borderline High 200 - 499 High >/=500 Very High	mg/dL
METHOD : ENZYMATIC ASSAY			
HDL CHOLESTEROL	55	< 40 Low >/=60 High	mg/dL
METHOD : DIRECT MEASURE - PEG			
LDL CHOLESTEROL, DIRECT	**159 High**	< 100 Optimal 100 - 129 Near or above optimal 130 - 159 Borderline High 160 - 189 High >/= 190 Very High	mg/dL
METHOD : DIRECT MEASURE WITHOUT SAMPLE PRETREATMENT			
NON HDL CHOLESTEROL	**187 High**	Desirable: Less than 130 Above Desirable: 130 - 159 Borderline High: 160 - 189 High: 190 - 219 Very high: > or = 220	mg/dL
METHOD : CALCULATED PARAMETER			
VERY LOW DENSITY LIPOPROTEIN	24.4	</= 30.0	mg/dL
METHOD : CALCULATED PARAMETER			
CHOL/HDL RATIO	4.4	3.3 - 4.4 Low Risk 4.5 - 7.0 Average Risk 7.1 - 11.0 Moderate Risk > 11.0 High Risk	
METHOD : CALCULATED PARAMETER			

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LDL/HDL RATIO	2.9	0.5 - 3.0 Desirable/Low Risk 3.1 - 6.0 Borderline/Moderate Risk >6.0 High Risk	

METHOD : CALCULATED PARAMETER

Interpretation(s)

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CLINICAL PATH - URINALYSIS

KIDNEY PANEL - 1

PHYSICAL EXAMINATION, URINE

COLOR	PALE YELLOW		
METHOD : PHYSICAL			
APPEARANCE	CLEAR		
METHOD : VISUAL			

CHEMICAL EXAMINATION, URINE

PH	7.0	4.7 - 7.5	
METHOD : REFLECTANCE SPECTROPHOTOMETRY- DOUBLE INDICATOR METHOD			
SPECIFIC GRAVITY	1.015	1.003 - 1.035	
METHOD : REFLECTANCE SPECTROPHOTOMETRY (APPARENT PKA CHANGE OF PRETREATED POLYELECTROLYTES IN RELATION TO IONIC CONCENTRATION)			
PROTEIN	NOT DETECTED	NOT DETECTED	
METHOD : REFLECTANCE SPECTROPHOTOMETRY - PROTEIN-ERROR-OF-INDICATOR PRINCIPLE			
GLUCOSE	NOT DETECTED	NOT DETECTED	
METHOD : REFLECTANCE SPECTROPHOTOMETRY, DOUBLE SEQUENTIAL ENZYME REACTION-GOD/POD			
KETONES	NOT DETECTED	NOT DETECTED	
METHOD : REFLECTANCE SPECTROPHOTOMETRY, ROTHERA'S PRINCIPLE			
BLOOD	NOT DETECTED	NOT DETECTED	
METHOD : REFLECTANCE SPECTROPHOTOMETRY, PEROXIDASE LIKE ACTIVITY OF HAEMOGLOBIN			
BILIRUBIN	NOT DETECTED	NOT DETECTED	
METHOD : REFLECTANCE SPECTROPHOTOMETRY, DIAZOTIZATION- COUPLING OF BILIRUBIN WITH DIAZOTIZED SALT			
UROBILINOGEN	NORMAL	NORMAL	
METHOD : REFLECTANCE SPECTROPHOTOMETRY (MODIFIED EHRLICH REACTION)			
NITRITE	NOT DETECTED	NOT DETECTED	
METHOD : REFLECTANCE SPECTROPHOTOMETRY, CONVERSION OF NITRATE TO NITRITE			
LEUKOCYTE ESTERASE	NOT DETECTED	NOT DETECTED	
METHOD : REFLECTANCE SPECTROPHOTOMETRY, ESTERASE HYDROLYSIS ACTIVITY			

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MICROSCOPIC EXAMINATION, URINE			
RED BLOOD CELLS	NOT DETECTED	NOT DETECTED	/HPF
METHOD : MICROSCOPIC EXAMINATION			
PUS CELL (WBC'S)	0-1	0-5	/HPF
METHOD : MICROSCOPIC EXAMINATION			
EPITHELIAL CELLS	0-1	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	
METHOD : MICROSCOPIC EXAMINATION			
REMARKS	URINARY MICROSCOPIC EXAMINATION DONE ON URINARY CENTRIFUGED SEDIMENT		

Interpretation(s)

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SPECIALISED CHEMISTRY - HORMONE			
THYROID PANEL, SERUM			
T3	96.1	80.0 - 200.0	ng/dL
METHOD : ELECTROCHEMILUMINESCENCE IMMUNOASSAY, COMPETITIVE PRINCIPLE			
T4	5.95	5.10 - 14.10	µg/dL
METHOD : ELECTROCHEMILUMINESCENCE IMMUNOASSAY, COMPETITIVE PRINCIPLE			
TSH (ULTRASENSITIVE)	1.750	0.270 - 4.200	µIU/mL
METHOD : ELECTROCHEMILUMINESCENCE,SANDWICH IMMUNOASSAY			

Interpretation(s)

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SPECIALISED CHEMISTRY - TUMOR MARKER

PROSTATE SPECIFIC ANTIGEN, SERUM

PROSTATE SPECIFIC ANTIGEN	0.650	0.0 - 2.0	ng/mL

METHOD : ELECTROCHEMILUMINESCENCE,SANDWICH IMMUNOASSAY

Interpretation(s)
PROSTATE SPECIFIC ANTIGEN, SERUM-- PSA is detected in the male patients with normal, benign hyperplastic and malignant prostate tissue and in patients with prostatitis.
- PSA is not detected (or detected at very low levels) in the patients without prostate tissue (because of radical prostatectomy or cystoprostatectomy) and also in the female patients.
- It a suitable marker for monitoring of patients with Prostate Cancer and it is better to be used in conjunction with other diagnostic procedures.
- Serial PSA levels can help determine the success of prostatectomy and the need for further treatment, such as radiation, endocrine or chemotherapy and useful in detecting residual disease and early recurrence of tumor.
- Elevated levels of PSA can be also observed in the patients with non-malignant diseases like Prostatitis and Benign Prostatic Hyperplasia.
- Specimens for total PSA assay should be obtained before biopsy, prostatectomy or prostatic massage, since manipulation of the prostate gland may lead to elevated PSA (false positive) levels persisting up to 3 weeks.
- As per American urological guidelines, PSA screening is recommended for early detection of Prostate cancer above the age of 40 years. Following Age specific reference range can be used as a guide lines.
- Measurement of total PSA alone may not clearly distinguish between benign prostatic hyperplasia (BPH) from cancer, this is especially true for the total PSA values between 4-10 ng/mL.
- Total PSA values determined on patient samples by different testing procedures cannot be directly compared with one another and could be the cause of erroneous medical interpretations. Recommended follow up on same platform as patient result can vary due to differences in assay method and reagent specificity.

References-
1. Burtis CA, Ashwood ER, Bruns DE. Teitz textbook of clinical chemistry and Molecular Diagnostics. 4th edition.
2. Williamson MA, Snyder LM. Wallach's interpretation of diagnostic tests. 9th edition.

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