



UHID	13059631	Date	29/03/2024	
Name	Mr. Rohit Saraswat	Sex	Male	Age 36
OPD	Ophthal 14	Health Check Up		

Ch → NO

Hx NO

Drug allergy: → Not known

Sys illness: → NO

Habit → NO

U.V. → R 6/18P
 → L 6/18P (B.H.)

R → -1.50 @ 6/6
 L → -1.50 @ 6/6

M → R N6
 → L N6

C.V.S.
 20-20mle
 20mi / 30mi
 20mi 30mi
 C feet

IOP → R → 14.8
 → L → 15.4

All good

Soft dry

①-①-①-①
 Hmcs



7387696540

(April 3rd)

UHID	13059631	Date	29/03/2024		
Name	Mr. Rohit Saraswat	Sex	Male	Age	36
OPD	Dental 12 (7597378600)	Health Check Up			

B.O.B.

Drug allergy:
 Sys illness:

M/H → N RH.

O/E → missing $\frac{+}{6}$

→ Impacted $\frac{+}{8}$ + pericoronitis $\frac{+}{8}$

→ grossly carious $\frac{8}{1}$

→ Impacted $\frac{1}{8}$

→ R.P $\frac{6}{+}$

→ stains ++, calculus +

Rx → Adv. scaling

→ Adv. OPG $\frac{8}{8}$ estn.

→ Adv estn followed by replacement $\frac{1}{6}$

→ Adv. replacement $\frac{6}{6}$

→ Adv CBCT.

Parsha
 Divya Shalilam
 MDS (Perio)
 A-391.07

PATIENT NAME : MR.ROHIT SARASWAT

REF. DOCTOR :

CODE/NAME & ADDRESS : C000045507

FORTIS VASHI-CHC -SPLZD
FORTIS HOSPITAL # VASHI,
MUMBAI 440001

ACCESSION NO : 0022XC006143

PATIENT ID : FH.13059631

CLIENT PATIENT ID: UID:13059631

ABHA NO :

AGE/SEX : 36 Years Male

DRAWN : 29/03/2024 09:24:00

RECEIVED : 29/03/2024 09:25:33

REPORTED : 29/03/2024 15:24:10

CLINICAL INFORMATION :

UID:13059631 REQNO-1684546

CORP-OPD

BILLNO-150124OPCR017781

BILLNO-150124OPCR017781

Test Report Status	Final	Results	Biological Reference Interval	Units
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HAEMATOLOGY - CBC

CBC-5, EDTA WHOLE BLOOD

BLOOD COUNTS, EDTA WHOLE BLOOD

HEMOGLOBIN (HB)	15.1	13.0 - 17.0	g/dL
METHOD : SLS METHOD			
RED BLOOD CELL (RBC) COUNT	5.42	4.5 - 5.5	mil/ μ L
METHOD : HYDRODYNAMIC FOCUSING			
WHITE BLOOD CELL (WBC) COUNT	8.67	4.0 - 10.0	thou/ μ L
METHOD : FLUORESCENCE FLOW CYTOMETRY			
PLATELET COUNT	305	150 - 410	thou/ μ L
METHOD : HYDRODYNAMIC FOCUSING BY DC DETECTION			

RBC AND PLATELET INDICES

HEMATOCRIT (PCV)	45.3	40.0 - 50.0	%
METHOD : CUMULATIVE PULSE HEIGHT DETECTION METHOD			
MEAN CORPUSCULAR VOLUME (MCV)	83.6	83.0 - 101.0	fL
METHOD : CALCULATED PARAMETER			
MEAN CORPUSCULAR HEMOGLOBIN (MCH)	27.9	27.0 - 32.0	pg
METHOD : CALCULATED PARAMETER			
MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION(MCHC)	33.3	31.5 - 34.5	g/dL
METHOD : CALCULATED PARAMETER			
RED CELL DISTRIBUTION WIDTH (RDW)	11.7	11.6 - 14.0	%
METHOD : CALCULATED PARAMETER			
MENTZER INDEX	15.4		
METHOD : CALCULATED PARAMETER			
MEAN PLATELET VOLUME (MPV)	9.7	6.8 - 10.9	fL
METHOD : CALCULATED PARAMETER			

WBC DIFFERENTIAL COUNT



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Consultant Pathologist

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Patient Ref. No. 22000000912050

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NEUTROPHILS		45	40.0 - 80.0	%
METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING				
LYMPHOCYTES		37	20.0 - 40.0	%
METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING				
MONOCYTES		5	2.0 - 10.0	%
METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING				
EOSINOPHILS		13 High	1 - 6	%
METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING				
BASOPHILS		0	0 - 2	%
METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING				
ABSOLUTE NEUTROPHIL COUNT		3.90	2.0 - 7.0	thou/ μ L
METHOD : CALCULATED PARAMETER				
ABSOLUTE LYMPHOCYTE COUNT		3.21 High	1.0 - 3.0	thou/ μ L
METHOD : CALCULATED PARAMETER				
ABSOLUTE MONOCYTE COUNT		0.43	0.2 - 1.0	thou/ μ L
METHOD : CALCULATED PARAMETER				
ABSOLUTE EOSINOPHIL COUNT		1.13 High	0.02 - 0.50	thou/ μ L
METHOD : CALCULATED PARAMETER				
ABSOLUTE BASOPHIL COUNT		0 Low	0.02 - 0.10	thou/ μ L
METHOD : CALCULATED PARAMETER				
NEUTROPHIL LYMPHOCYTE RATIO (NLR)		1.2		
METHOD : CALCULATED				

MORPHOLOGY

RBC

METHOD : MICROSCOPIC EXAMINATION

PREDOMINANTLY NORMOCYTIC NORMOCHROMIC

WBC

METHOD : MICROSCOPIC EXAMINATION

EOSINOPHILIA PRESENT

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.



Dr. Akshay Dhotre, MD
(Reg.no. MMC 2019/09/6377)
Consultant Pathologist

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HAEMATOLOGY

ERYTHROCYTE SEDIMENTATION RATE (ESR), EDTA BLOOD

E.S.R 06 0 - 14 mm at 1 hr
METHOD : WESTERGRÉN METHOD

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 : HB VARIANT (HPLC)

ESTIMATED AVERAGE GLUCOSE(EAG) 114.0 < 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, Vasculitides, 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: 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)



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Consultant Pathologist

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Test Report Status **Final**

Results

Biological Reference Interval Units

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 patient's metabolic control has remained continuously within the target range.
1. eAG (Estimated average glucose) converts percentage HbA1c to mg/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 platform (Boronate affinity chromatography) is recommended for testing of HbA1c. Abnormal Hemoglobin electrophoresis (HPLC method) is recommended for detecting a hemoglobinopathy



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(Reg, no. MMC 2019/09/6377)
Consultant Pathologist

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IMMUNOHAEMATOLOGY

ABO GROUP & RH TYPE, EDTA WHOLE BLOOD

ABO GROUP	TYPE AB
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	0.54	0.2 - 1.0	mg/dL
METHOD : JENDRASSIK AND GROFF			
BILIRUBIN, DIRECT	0.13	0.0 - 0.2	mg/dL
METHOD : JENDRASSIK AND GROFF			
BILIRUBIN, INDIRECT	0.41	0.1 - 1.0	mg/dL
METHOD : CALCULATED PARAMETER			
TOTAL PROTEIN	7.9	6.4 - 8.2	g/dL
METHOD : BIURET			
ALBUMIN	4.5	3.4 - 5.0	g/dL
METHOD : BCP DYE BINDING			
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)	23	15 - 37	U/L
METHOD : UV WITH P5P			
ALANINE AMINOTRANSFERASE (ALT/SGPT)	51 High	< 45.0	U/L
METHOD : UV WITH P5P			
ALKALINE PHOSPHATASE	49	30 - 120	U/L
METHOD : PNPP-ANP			
GAMMA GLUTAMYL TRANSFERASE (GGT)	41	15 - 85	U/L
METHOD : GAMMA GLUTAMYL CARBOXY 4NITROANILIDE			
LACTATE DEHYDROGENASE	160	85 - 227	U/L
METHOD : LACTATE -PYRUVATE			

GLUCOSE FASTING,FLUORIDE PLASMA

FBS (FASTING BLOOD SUGAR)	99	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 11 6 - 20 mg/dL
 METHOD : UREASE - UV

CREATININE EGFR- EPI

CREATININE 0.95 0.90 - 1.30 mg/dL
 METHOD : ALKALINE PICRATE KINETIC JAFFES

AGE 36 years

GLOMERULAR FILTRATION RATE (MALE) 106.38 Refer Interpretation Below mL/min/1.73m2
 METHOD : CALCULATED PARAMETER

BUN/CREAT RATIO

BUN/CREAT RATIO 11.58 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 7.9 6.4 - 8.2 g/dL
 METHOD : BIURET

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ALBUMIN, SERUM

ALBUMIN	4.5	3.4 - 5.0	g/dL
METHOD : BCP DYE BINDING			

GLOBULIN

GLOBULIN	3.4	2.0 - 4.1	g/dL
METHOD : CALCULATED PARAMETER			

ELECTROLYTES (NA/K/CL), SERUM

SODIUM, SERUM	140	136 - 145	mmol/L
METHOD : ISE INDIRECT			
POTASSIUM, SERUM	4.37	3.50 - 5.10	mmol/L
METHOD : ISE INDIRECT			
CHLORIDE, SERUM	102	98 - 107	mmol/L
METHOD : ISE INDIRECT			

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.

Dr. Akshay Dhotre, MD
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Patient Ref. No. 22000000912050

PATIENT NAME : MR.ROHIT SARASWAT

REF. DOCTOR :

CODE/NAME & ADDRESS : C000045507
 FORTIS VASHI-CHC -SPLZD
 FORTIS HOSPITAL # VASHI,
 MUMBAI 440001

ACCESSION NO : 0022XC006143
PATIENT ID : FH.13059631
CLIENT PATIENT ID: UID:13059631
ABHA NO :

AGE/SEX : 36 Years Male
DRAWN : 29/03/2024 09:24:00
RECEIVED : 29/03/2024 09:25:33
REPORTED : 29/03/2024 15:24:10

CLINICAL INFORMATION :

UID:13059631 REQNO-1684546
 CORP-OPD
 BILLNO-150124OPCR017781
 BILLNO-150124OPCR017781

Test Report Status	Final	Results	Biological Reference Interval	Units
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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 so that 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, sulfonureas, 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 Glycosuria, 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 (KDIGO) 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, Lasch 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.

Dr. Akshay Dhotre, MD
 (Reg.no. MMC 2019/09/6377)
 Consultant Pathologist



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PERFORMED AT :

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 Navi Mumbai, 400703
 Maharashtra, India
 Tel : 022-39199222, 022-49723322, Fax :
 CIN - U74899PB1995PLC045956
 Email : -



Patient Ref. No. 2200000912050

PATIENT NAME : MR.ROHIT SARASWAT		REF. DOCTOR :	
CODE/NAME & ADDRESS : C000045507 FORTIS VASHI-CHC -SPLZD FORTIS HOSPITAL # VASHI, MUMBAI 440001	ACCESSION NO : 0022XC006143 PATIENT ID : FH.13059631 CLIENT PATIENT ID: UID:13059631 ABHA NO :	AGE/SEX : 36 Years Male DRAWN : 29/03/2024 09:24:00 RECEIVED : 29/03/2024 09:25:33 REPORTED : 29/03/2024 15:24:10	

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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.

Dr. Akshay Dhotre, MD
 (Reg.no. MMC 2019/09/6377)
 Consultant Pathologist



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 Hiranandani Hospital-Vashi, Mini Seashore Road, Sector 10,
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BIOCHEMISTRY - LIPID

LIPID PROFILE, SERUM

CHOLESTEROL, TOTAL 161 < 200 Desirable mg/dL
 200 - 239 Borderline High
 >/= 240 High

METHOD : ENZYMATIC/COLORIMETRIC, CHOLESTEROL OXIDASE, ESTERASE, PEROXIDASE

TRIGLYCERIDES 271 High < 150 Normal mg/dL
 150 - 199 Borderline High
 200 - 499 High
 >/=500 Very High

METHOD : ENZYMATIC ASSAY

HDL CHOLESTEROL 37 Low < 40 Low mg/dL
 >/=60 High

METHOD : DIRECT MEASURE - PEG

LDL CHOLESTEROL, DIRECT 87 < 100 Optimal mg/dL
 100 - 129 Near or above optimal
 130 - 159 Borderline High
 160 - 189 High
 >/= 190 Very High

METHOD : DIRECT MEASURE WITHOUT SAMPLE PRETREATMENT

NON HDL CHOLESTEROL 124 Desirable: Less than 130 mg/dL
 Above Desirable: 130 - 159
 Borderline High: 160 - 189
 High: 190 - 219
 Very high: > or = 220

METHOD : CALCULATED PARAMETER

VERY LOW DENSITY LIPOPROTEIN 54.2 High </= 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

Dr. Akshay Dhotre, MD
 (Reg,no. MMC 2019/09/6377)
 Consultant Pathologist



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LDL/HDL RATIO		2.4	0.5 - 3.0 Desirable/Low Risk 3.1 - 6.0 Borderline/Moderate Risk >6.0 High Risk	
---------------	--	-----	--	--

METHOD : CALCULATED PARAMETER

Interpretation(s)

Dr. Akshay Dhotre, MD
(Reg,no. MMC 2019/09/6377)
Consultant Pathologist



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Hiranandani Hospital-Vashi, Mini Seashore Road, Sector 10,
Navi Mumbai, 400703
Maharashtra, India
Tel : 022-39199222,022-49723322, Fax :
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Patient Ref. No. 22000000912050

PATIENT NAME : MR.ROHIT SARASWAT		REF. DOCTOR :
CODE/NAME & ADDRESS : C000045507	ACCESSION NO : 0022XC006143	AGE/SEX : 36 Years Male
FORTIS VASHI-CHC -SPLZD	PATIENT ID : FH.13059631	DRAWN : 29/03/2024 09:24:00
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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	7.5	4.7 - 7.5
METHOD : REFLECTANCE SPECTROPHOTOMETRY- DOUBLE INDICATOR METHOD		
SPECIFIC GRAVITY	1.020	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		

Dr. Akshay Dhotre, MD
 (Reg.no. MMC 2019/09/6377)
 Consultant Pathologist

Dr. Rekha Nair, MD
 (Reg No. MMC 2001/06/2354)
 Microbiologist



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REF. DOCTOR :

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 FORTIS HOSPITAL # VASHI,
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ACCESSION NO : 0022XC006143
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MICROSCOPIC EXAMINATION, URINE

RED BLOOD CELLS METHOD : MICROSCOPIC EXAMINATION	NOT DETECTED	NOT DETECTED	/HPF
PUS CELL (WBC'S) METHOD : MICROSCOPIC EXAMINATION	0-1	0-5	/HPF
EPITHELIAL CELLS METHOD : MICROSCOPIC EXAMINATION	0-1	0-5	/HPF
CASTS METHOD : MICROSCOPIC EXAMINATION	NOT DETECTED		
CRYSTALS METHOD : MICROSCOPIC EXAMINATION	NOT DETECTED		
BACTERIA METHOD : MICROSCOPIC EXAMINATION	NOT DETECTED	NOT DETECTED	
YEAST METHOD : MICROSCOPIC EXAMINATION	NOT DETECTED	NOT DETECTED	

REMARKS URINARY MICROSCOPIC EXAMINATION DONE ON URINARY CENTRIFUGED SEDIMENT

Interpretation(s)

Dr. Akshay Dhotre, MD
 (Reg.no. MMC 2019/09/6377)
 Consultant Pathologist

Dr. Rekha Nair, MD
 (Reg No. MMC 2001/06/2354)
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PATIENT NAME : MR.ROHIT SARASWAT

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ACCESSION NO : **0022XC006143**

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CLIENT PATIENT ID: UID:13059631

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SPECIALISED CHEMISTRY - HORMONE

THYROID PANEL, SERUM

T3	159.9	80.0 - 200.0	ng/dL
METHOD : ELECTROCHEMILUMINESCENCE IMMUNOASSAY, COMPETITIVE PRINCIPLE			
T4	8.96	5.10 - 14.10	µg/dL
METHOD : ELECTROCHEMILUMINESCENCE IMMUNOASSAY, COMPETITIVE PRINCIPLE			
TSH (ULTRASENSITIVE)	2.270	0.270 - 4.200	µIU/mL
METHOD : ELECTROCHEMILUMINESCENCE,SANDWICH IMMUNOASSAY			

Interpretation(s)

Dr. Akshay Dhotre, MD
 (Reg.no. MMC 2019/09/6377)
 Consultant Pathologist



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Test Report Status **Final**

Results

Biological Reference Interval Units

SPECIALISED CHEMISTRY - TUMOR MARKER

PROSTATE SPECIFIC ANTIGEN, SERUM

PROSTATE SPECIFIC ANTIGEN 0.743 0.0 - 1.4 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.

End Of Report

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Dr. Akshay Dhotre, MD
(Reg.no. MMC 2019/09/6377)
Consultant Pathologist



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ACCESSION NO : 0022XC006199

PATIENT ID : FH.13059631

CLIENT PATIENT ID: UID:13059631

ABHA NO :

AGE/SEX : 36 Years Male

DRAWN : 29/03/2024 11:53:00

RECEIVED : 29/03/2024 11:55:02

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CLINICAL INFORMATION :

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BIOCHEMISTRY

GLUCOSE, POST-PRANDIAL, PLASMA

PPBS(POST PRANDIAL BLOOD SUGAR)	100	70 - 140	mg/dL
METHOD : HEXOKINASE			

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 Glycosuria, Glycaemic index & response to food consumed, Alimentary Hypoglycemia, Increased insulin response & sensitivity etc.Additional test HbA1c

****End Of Report****

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Patient Ref. No. 22000000912106

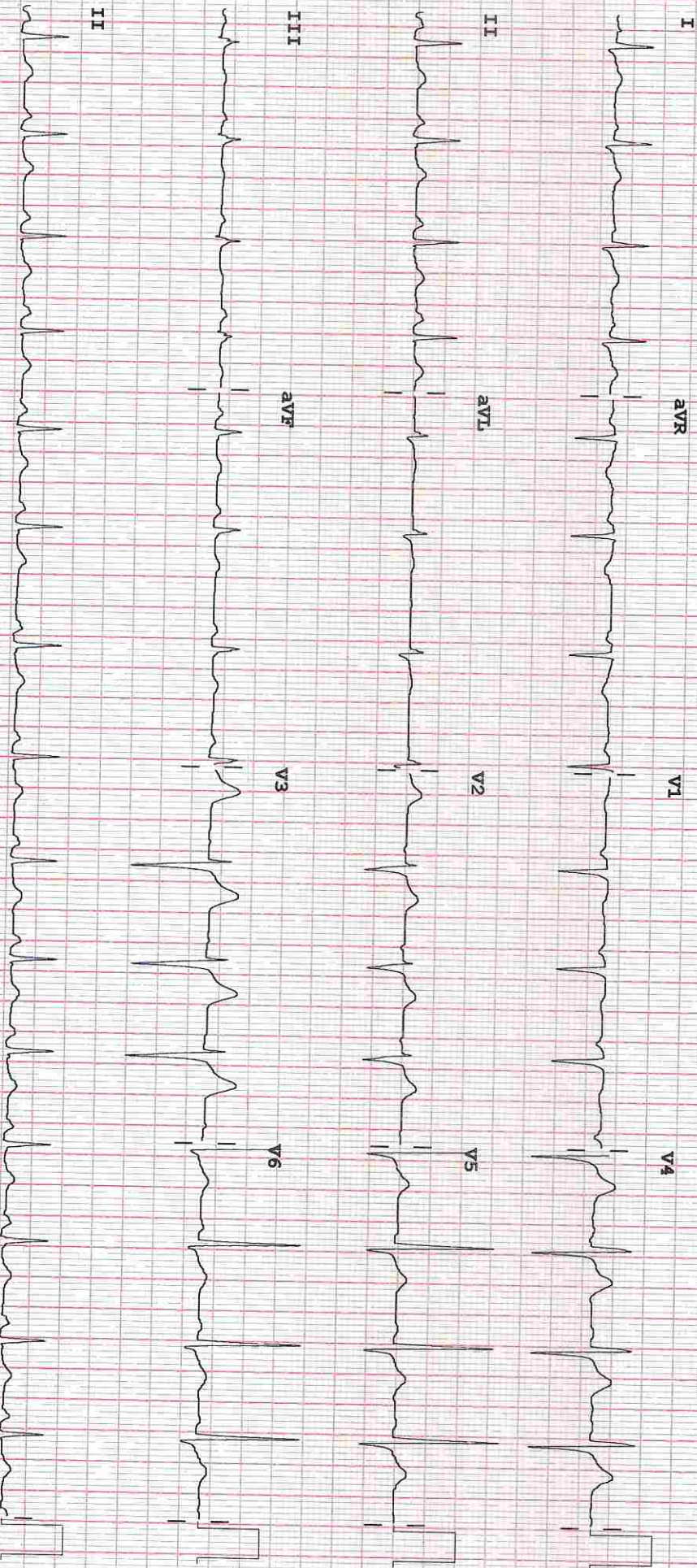
Rate 92 Sinus rhythm
 PR 140 Probable left atrial enlargement
 QRSD 86
 QT 346
 QTc 429

--AXIS--
 P 63
 QRS 58
 T 47

12 Lead; Standard Placement

- BORDERLINE ECG -

Unconfirmed Diagnosis



Device:

Speed: 25 mm/sec

Limb: 10 mm/mV

Chest: 10.0 mm/mV

F 50~ 0.50-100 Hz W

100B CL

p2

Normal

HL



(For Billing/Reports & Discharge Summary only)

DEPARTMENT OF NIC

Date: 29/Mar/2024

Name: Mr. Rohit Saraswat
Age | Sex: 36 YEAR(S) | Male
Order Station : FO-OPD
Bed Name :

UHID | Episode No : 13059631 | 18048/24/1501
Order No | Order Date: 1501/PN/OP/2403/37756 | 29-Mar-2024
Admitted On | Reporting Date : 29-Mar-2024 15:17:59
Order Doctor Name : Dr.SELF .

TREAD MILL TEST (TMT)

Resting Heart rate	107 bpm
Resting Blood pressure	120/80 mmHg
Medication	Nil
Supine ECG	Normal
Standard protocol	BRUCE
Total Exercise time	7 min 27 seconds
Maximum heart rate	160 bpm
Maximum blood pressure	130/80 mmHg
Workload achieved	10.10 METS
Reason for termination	Target heart rate achieved

Final Impression :

STRESS TEST IS NEGATIVE FOR EXERCISE INDUCED MYOCARDIAL ISCHEMIA AT 10.10 METS AND 86 % OF MAXIMUM PREDICTED HEART RATE.


DR.PRASHANT PAWAR,
DNB(MED),DNB(CARD)

DR.AMIT SINGH,
MD(MED), DM(CARD)

Hiranandani Healthcare Pvt. Ltd.

Mini Sea Shore Road, Sector 10-A, Vashi, Navi Mumbai - 400703.

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CIN: U85100MH2005PTC 154823

GST IN : 27AABCH5894D1ZG

PAN NO : AABCH5894D



Hiranandani
HOSPITAL

(A Fortis Network Hospital)

DEPARTMENT OF RADIOLOGY

Date: 29/Mar/2024

Name: Mr. Rohit Saraswat

Age | Sex: 36 YEAR(S) | Male

Order Station : FO-OPD

Bed Name :

UHID | Episode No : 13059631 | 18048/24/1501

Order No | Order Date: 1501/PN/OP/2403/37756 | 29-Mar-2024

Admitted On | Reporting Date : 29-Mar-2024 11:11:20

Order Doctor Name : Dr.SELF .

X-RAY-CHEST- PA

Findings:

Both lung fields are clear.

The cardiac shadow appears within normal limits.

Trachea and major bronchi appears normal.

Both costophrenic angles are well maintained.

Bony thorax is unremarkable.

DR. YOGINI SHAH
DMRD., DNB. (Radiologist)



(For Billing/Reports & Discharge Summary only)

Patient Name	: Rohit Saraswat	Patient ID	: 13059631
Sex / Age	: M / 36Y 4M 26D	Accession No.	: PHC.7813656
Modality	: US	Scan DateTime	: 29-03-2024 11:15:49
IPID No	: 18048/24/1501	ReportDatetime	: 29-03-2024 12:13:43

USG - WHOLE ABDOMEN

LIVER is normal in size and shows moderately raised echogenicity. No IHBR dilatation. No focal lesion is seen in liver. Portal vein appears normal in caliber.

GALL BLADDER is physiologically distended. Gall bladder reveals normal wall thickness. No evidence of calculi in gall bladder. No evidence of pericholecystic collection.

CBD appears normal in caliber.

SPLEEN is normal in size and echogenicity.

BOTH KIDNEYS are normal in size and echogenicity. The central sinus complex is normal. No evidence of calculi/hydronephrosis.

Right kidney measures 10.5 x 4.4 cm.

Left kidney measures 10.3 x 4.9 cm.

PANCREAS : Head and body of pancreas is unremarkable. Rest of the pancreas is obscured.

URINARY BLADDER is normal in capacity and contour. Bladder wall is normal in thickness. No evidence of intravesical calculi.

PROSTATE is normal in size & echogenicity. It measures ~ 18.9 cc in volume.

No evidence of ascites.

Impression:

- **Grade II fatty infiltration of liver.**

DR. KUNAL NIGAM
M.D. (Radiologist)