



BMI CHART

Date: 13/01/24

Name: Renu Patel

Age: 41 yrs

Sex: M / F

BP: _____ Height (cms): _____ Weight (kgs): _____ BMI: _____

WEIGHT lbs kgs	100		115		130		145		160		175		190		205		210		215																																									
	45.5	47.7	50.5	52.3	54.8	56.9	59.1	61.4	63.6	65.9	68.2	70.5	72.7	75.0	77.3	79.5	81.8	84.1	86.4	88.6	90.9	93.2	95.5	97.7																																				
HEIGHT in/cm	Underweight												Healthy												Overweight												Obese												Extremely Obese											
	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38	39	40	41	42																																				
5'0" - 152.4	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38	39	40	41	42																																				
5'1" - 154.9	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38	39	40	41																																				
5'2" - 157.4	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38	39	40	41																																				
5'3" - 160.0	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38	39	40																																				
5'4" - 162.5	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38	39	40																																				
5'5" - 165.1	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38	39																																				
5'6" - 167.6	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38	39																																				
5'7" - 170.1	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38																																				
5'8" - 172.7	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38																																				
5'9" - 175.2	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37																																				
5'10" - 177.8	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37																																				
5'11" - 180.3	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37																																				
6'0" - 182.8	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36																																				
6'1" - 185.4	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36																																				
6'2" - 187.9	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35																																				
6'3" - 190.5	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35																																				
6'4" - 193.0	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35																																				

Doctors Notes:

Signature

PAP



PATIENT NAME : MRS.RANU PATEL		REF. DOCTOR :	
CODE/NAME & ADDRESS : C00045507 FORTIS VASHI-CHC -SPLZD FORTIS HOSPITAL # VASHI, MUMBAI 440001		ACCESSION NO : 0022XA002426 PATIENT ID : FH.12921812 CLIENT PATIENT ID: UID:12921812 ABHA NO :	AGE/SEX : 41 Years Female DRAWN : 13/01/2024 12:49:00 RECEIVED : 13/01/2024 12:49:24 REPORTED : 13/01/2024 14:28:14

CLINICAL INFORMATION :

UID:12921812 REQNO-1650220
 CORP-OPD
 BILLNO-150124OPCR002520
 BILLNO-150124OPCR002520

Test Report Status	Results	Biological Reference Interval	Units
Final			

BIOCHEMISTRY

GLUCOSE, POST-PRANDIAL, PLASMA

PPBS(POST PRANDIAL BLOOD SUGAR)	75	70 - 140	mg/dL
METHOD : HEXOKINASE			

Comments

NOTE: - 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 Glycosuria, 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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AGE/SEX : 41 Years Female
 DRAWN : 13/01/2024 10:28:00
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HAEMATOLOGY - CBC

CBC-5, EDTA WHOLE BLOOD

BLOOD COUNTS, EDTA WHOLE BLOOD

HEMOGLOBIN (HB)	12.1	12.0 - 15.0	g/dL
METHOD : SLS METHOD			
RED BLOOD CELL (RBC) COUNT	4.34	3.8 - 4.8	mil/ μ L
METHOD : HYDRODYNAMIC FOCUSING			
WHITE BLOOD CELL (WBC) COUNT	5.40	4.0 - 10.0	thou/ μ L
METHOD : FLUORESCENCE FLOW CYTOMETRY			
PLATELET COUNT	175	150 - 410	thou/ μ L
METHOD : HYDRODYNAMIC FOCUSING BY DC DETECTION			

RBC AND PLATELET INDICES

HEMATOCRIT (PCV)	35.6 Low	36.0 - 46.0	%
METHOD : CUMULATIVE PULSE HEIGHT DETECTION METHOD			
MEAN CORPUSCULAR VOLUME (MCV)	82.0 Low	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)	34.0	31.5 - 34.5	g/dL
METHOD : CALCULATED PARAMETER			
RED CELL DISTRIBUTION WIDTH (RDW)	13.1	11.6 - 14.0	%
METHOD : CALCULATED PARAMETER			
MENTZER INDEX	18.9		
METHOD : CALCULATED PARAMETER			
MEAN PLATELET VOLUME (MPV)	11.7 High	6.8 - 10.9	fL
METHOD : CALCULATED PARAMETER			

WBC DIFFERENTIAL COUNT



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NEUTROPHILS		66	40.0 - 80.0	%
METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING				
LYMPHOCYTES		24	20.0 - 40.0	%
METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING				
MONOCYTES		8	2.0 - 10.0	%
METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING				
EOSINOPHILS		2	1 - 6	%
METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING				
BASOPHILS		0	0 - 2	%
METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING				
ABSOLUTE NEUTROPHIL COUNT		3.56	2.0 - 7.0	thou/ μ L
METHOD : CALCULATED PARAMETER				
ABSOLUTE LYMPHOCYTE COUNT		1.30	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		0.11	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)		2.7		
METHOD : CALCULATED				

MORPHOLOGY

RBC PREDOMINANTLY NORMOCYTIC NORMOCHROMIC
 METHOD : MICROSCOPIC EXAMINATION

WBC NORMAL MORPHOLOGY
 METHOD : MICROSCOPIC EXAMINATION

PLATELETS ADEQUATE
 METHOD : MICROSCOPIC EXAMINATION

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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), EDTA BLOOD

E.S.R 11 0 - 20 mm at 1 hr
METHOD : WESTERGREN METHOD

GLYCOSYLATED HEMOGLOBIN(HBA1C), EDTA WHOLE BLOOD

HBA1C 4.8 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) 91.1 < 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 0-40 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, (Sickle Cells, 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:

- Evaluating the long-term control of blood glucose concentrations in diabetic patients.
- Diagnosing diabetes.
- 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.

- eAG (Estimated average glucose) converts percentage HbA1c to mg/dl, to compare blood glucose levels.
- eAG gives an evaluation of blood glucose levels for the last couple of months.
- eAG is calculated as $eAG (mg/dl) = 28.7 * HbA1c - 46.7$

HbA1c Estimation can get affected due to :

- 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.
- Vitamin C & E are reported to falsely lower test results (possibly by inhibiting glycation of hemoglobin).
- 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.
- Interference of hemoglobinopathies in HbA1c estimation is seen in

- Homozygous hemoglobinopathy. Fructosamine is recommended for testing of HbA1c.
- Heterozygous state detected (D10 is corrected for HbS & HbC trait.)
- 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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IMMUNOHAEMATOLOGY

ABO GROUP & RH TYPE, EDTA WHOLE BLOOD

ABO GROUP	TYPE B
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	0.57	0.2 - 1.0	mg/dL
METHOD : JENDRASSIK AND GROFF			
BILIRUBIN, DIRECT	0.19	0.0 - 0.2	mg/dL
METHOD : JENDRASSIK AND GROFF			
BILIRUBIN, INDIRECT	0.38	0.1 - 1.0	mg/dL
METHOD : CALCULATED PARAMETER			
TOTAL PROTEIN	7.5	6.4 - 8.2	g/dL
METHOD : BIURET			
ALBUMIN	3.7	3.4 - 5.0	g/dL
METHOD : BCP DYE BINDING			
GLOBULIN	3.8	2.0 - 4.1	g/dL
METHOD : CALCULATED PARAMETER			
ALBUMIN/GLOBULIN RATIO	1.0	1.0 - 2.1	RATIO
METHOD : CALCULATED PARAMETER			
ASPARTATE AMINOTRANSFERASE(AST/SGOT)	11 Low	15 - 37	U/L
METHOD : UV WITH P5P			
ALANINE AMINOTRANSFERASE (ALT/SGPT)	16	< 34.0	U/L
METHOD : UV WITH P5P			
ALKALINE PHOSPHATASE	68	30 - 120	U/L
METHOD : PNPP-ANP			
GAMMA GLUTAMYL TRANSFERASE (GGT)	16	5 - 55	U/L
METHOD : GAMMA GLUTAMYL CARBOXY 4NITROANILIDE			
LACTATE DEHYDROGENASE	133	81 - 234	U/L
METHOD : LACTATE -PYRUVATE			

GLUCOSE FASTING, FLUORIDE PLASMA

FBS (FASTING BLOOD SUGAR)	92	Normal : < 100 Pre-diabetes: 100-125 Diabetes: >=126	mg/dL
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METHOD : HEXOKINASE



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RECEIVED : 13/01/2024 10:28:35
REPORTED : 13/01/2024 16:10:07

CLINICAL INFORMATION :

UID:12921812 REQNO-1650220
 CORP-OPD
 BILLNO-150124OPCR002520
 BILLNO-150124OPCR002520

Test Report Status	Final	Results	Biological Reference Interval	Units
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KIDNEY PANEL - 1

BLOOD UREA NITROGEN (BUN), SERUM

BLOOD UREA NITROGEN	6	6 - 20	mg/dL
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METHOD : UREASE - UV

CREATININE EGFR- EPI

CREATININE	0.77	0.60 - 1.10	mg/dL
------------	------	-------------	-------

METHOD : ALKALINE PICRATE KINETIC JAFFES

AGE	41		years
-----	----	--	-------

GLOMERULAR FILTRATION RATE (FEMALE)	99.32	Refer Interpretation Below	mL/min/1.73m ²
-------------------------------------	-------	----------------------------	---------------------------

METHOD : CALCULATED PARAMETER

BUN/CREAT RATIO

BUN/CREAT RATIO	7.79	5.00 - 15.00	
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METHOD : CALCULATED PARAMETER

URIC ACID, SERUM

URIC ACID	3.9	2.6 - 6.0	mg/dL
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METHOD : URICASE UV

TOTAL PROTEIN, SERUM

TOTAL PROTEIN	7.5	6.4 - 8.2	g/dL
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METHOD : BIURET

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 CIN - U74899PB1995PLC045956
 Email :-



Patient Ref. No. 22000000896391

PATIENT NAME : MRS.RANU PATEL		REF. DOCTOR :	
CODE/NAME & ADDRESS : C000045507		ACCESSION NO : 0022XA002363	AGE/SEX : 41 Years Female
FORTIS VASHI-CHC -SPLZD		PATIENT ID : FH.12921812	DRAWN : 13/01/2024 10:28:00
FORTIS HOSPITAL # VASHI,		CLIENT PATIENT ID: UID:12921812	RECEIVED : 13/01/2024 10:28:35
MUMBAI 440001		ABHA NO :	REPORTED : 13/01/2024 16:10:07

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ALBUMIN, SERUM				
ALBUMIN		3.7	3.4 - 5.0	g/dL
METHOD : BCP DYE BINDING				
GLOBULIN				
GLOBULIN		3.8	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		4.31	3.50 - 5.10	mmol/L
METHOD : ISE INDIRECT				
CHLORIDE, SERUM		105	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.

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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, 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 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, 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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Patient Ref. No. 2200000896391

PATIENT NAME : MRS.RANU PATEL

REF. DOCTOR :

CODE/NAME & ADDRESS : C000045507

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

ACCESSION NO : 0022XA002363

PATIENT ID : FH.12921812

CLIENT PATIENT ID: UID:12921812

ABHA NO :

AGE/SEX :41 Years Female

DRAWN :13/01/2024 10:28:00

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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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Patient Ref. No. 2200000896391

PATIENT NAME : MRS.RANU PATEL

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BIOCHEMISTRY - LIPID

LIPID PROFILE, SERUM

CHOLESTEROL, TOTAL	102	< 200 Desirable 200 - 239 Borderline High >= 240 High	mg/dL
METHOD : ENZYMATIC/COLORIMETRIC, CHOLESTEROL OXIDASE, ESTERASE, PEROXIDASE			
TRIGLYCERIDES	35	< 150 Normal 150 - 199 Borderline High 200 - 499 High >= 500 Very High	mg/dL
METHOD : ENZYMATIC ASSAY			
HDL CHOLESTEROL	40	< 40 Low >= 60 High	mg/dL
METHOD : DIRECT MEASURE - PEG			
LDL CHOLESTEROL, DIRECT	56	< 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	62	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	7.0	<= 30.0	mg/dL
METHOD : CALCULATED PARAMETER			
CHOL/HDL RATIO	2.6 Low	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	1.4	0.5 - 3.0 Desirable/Low Risk 3.1 - 6.0 Borderline/Moderate Risk >6.0 High Risk		
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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	6.0	4.7 - 7.5
METHOD : REFLECTANCE SPECTROPHOTOMETRY- DOUBLE INDICATOR METHOD		
SPECIFIC GRAVITY	<=1.005	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 METHOD : MICROSCOPIC EXAMINATION	NOT DETECTED	NOT DETECTED	/HPF
PUS CELL (WBC'S) METHOD : MICROSCOPIC EXAMINATION	2-3	0-5	/HPF
EPITHELIAL CELLS METHOD : MICROSCOPIC EXAMINATION	3-5	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

NOTE :- URINARY MICROSCOPIC EXAMINATION DONE ON URINARY CENTRIFUGED SEDIMENT

Interpretation(s)

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SPECIALISED CHEMISTRY - HORMONE

THYROID PANEL, SERUM

T3	146.7	Non-Pregnant Women 80.0 - 200.0 Pregnant Women 1st Trimester:105.0 - 230.0 2nd Trimester:129.0 - 262.0 3rd Trimester:135.0 - 262.0	ng/dL
METHOD : ELECTROCHEMILUMINESCENCE IMMUNOASSAY, COMPETITIVE PRINCIPLE			
T4	9.97	Non-Pregnant Women 5.10 - 14.10 Pregnant Women 1st Trimester: 7.33 - 14.80 2nd Trimester: 7.93 - 16.10 3rd Trimester: 6.95 - 15.70	µg/dL
METHOD : ELECTROCHEMILUMINESCENCE IMMUNOASSAY, COMPETITIVE PRINCIPLE			
TSH (ULTRASENSITIVE)	0.945	Non Pregnant Women 0.27 - 4.20 Pregnant Women (As per American Thyroid Association) 1st Trimester 0.100 - 2.500 2nd Trimester 0.200 - 3.000 3rd Trimester 0.300 - 3.000	µIU/mL
METHOD : ELECTROCHEMILUMINESCENCE SANDWICH IMMUNOASSAY			

Interpretation(s)

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FORTIS VASHI-CHC -SPLZD

FORTIS HOSPITAL # VASHI, (

MUMBAI 440001

ACCESSION NO : 0022XA002454

PATIENT ID : FH.12921812

CLIENT PATIENT ID: UID:12921812

ABHA NO :

AGE/SEX :41 Years Female

DRAWN :13/01/2024 15:04:00

RECEIVED :13/01/2024 15:18:59

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CLINICAL INFORMATION :

UID:12921812 REQNO-1650220

CORP-OPD

BILLNO-150124OPCR002520

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Test Report Status **Final**

Units

CYTOLOGY

PAPANICOLAOU SMEAR

PAPANICOLAOU SMEAR

TEST METHOD

SPECIMEN TYPE

REPORTING SYSTEM

SPECIMEN ADEQUACY

METHOD : MICROSCOPIC EXAMINATION

MICROSCOPY

CONVENTIONAL GYNEC CYTOLOGY

TWO UNSTAINED CERVICAL SMEARS RECEIVED

2014 BETHESDA SYSTEM FOR REPORTING CERVICAL CYTOLOGY

SATISFACTORY

SMEARS STUDIED SHOW SUPERFICIAL SQUAMOUS CELLS,
INTERMEDIATE SQUAMOUS CELLS, OCCASIONAL SQUAMOUS
METAPLASTIC CELLS, OCCASIONAL CLUSTERS OF ENDOCERVICAL CELLS
IN THE BACKGROUND OF FEW POLYMORPHS.

INTERPRETATION / RESULT

NEGATIVE FOR INTRAEPITHELIAL LESION OR MALIGNANCY

Comments

PLEASE NOTE PAPANICOLAOU SMEAR STUDY IS A SCREENING PROCEDURE FOR CERVICAL
CANCER WITH INHERENT FALSE NEGATIVE RESULTS, HENCE SHOULD BE INTERPRETED
WITH CAUTION.

NO CYTOLOGICAL EVIDENCE OF HPV INFECTION IN THE SMEARS STUDIED.

End Of Report

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



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

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View Details



View Report

PERFORMED AT :

Agilus Diagnostics Ltd.
Hiranandani Hospital-Vashi, Mini Seashore Road, Sector 10,
Navi Mumbai, 400703
Maharashtra, India
Tel : 022-39199222,022-49723322,
CIN - U74899PB1995PLC045956
Email : -



Patient Ref. No. 22000000896482

1/13/2024 11:40:02 AM

RANU PATEL
Female

12921812
41 Years

Rate 60 Sinus rhythm.normal P axis, V-rate 50-99
Borderline T abnormalities, inferior leads.T flat/neg, II III aVF

PR 139
QRS 83
QT 361
QTc 361

Handwritten:
V III, aVF
Complete
Circumflex

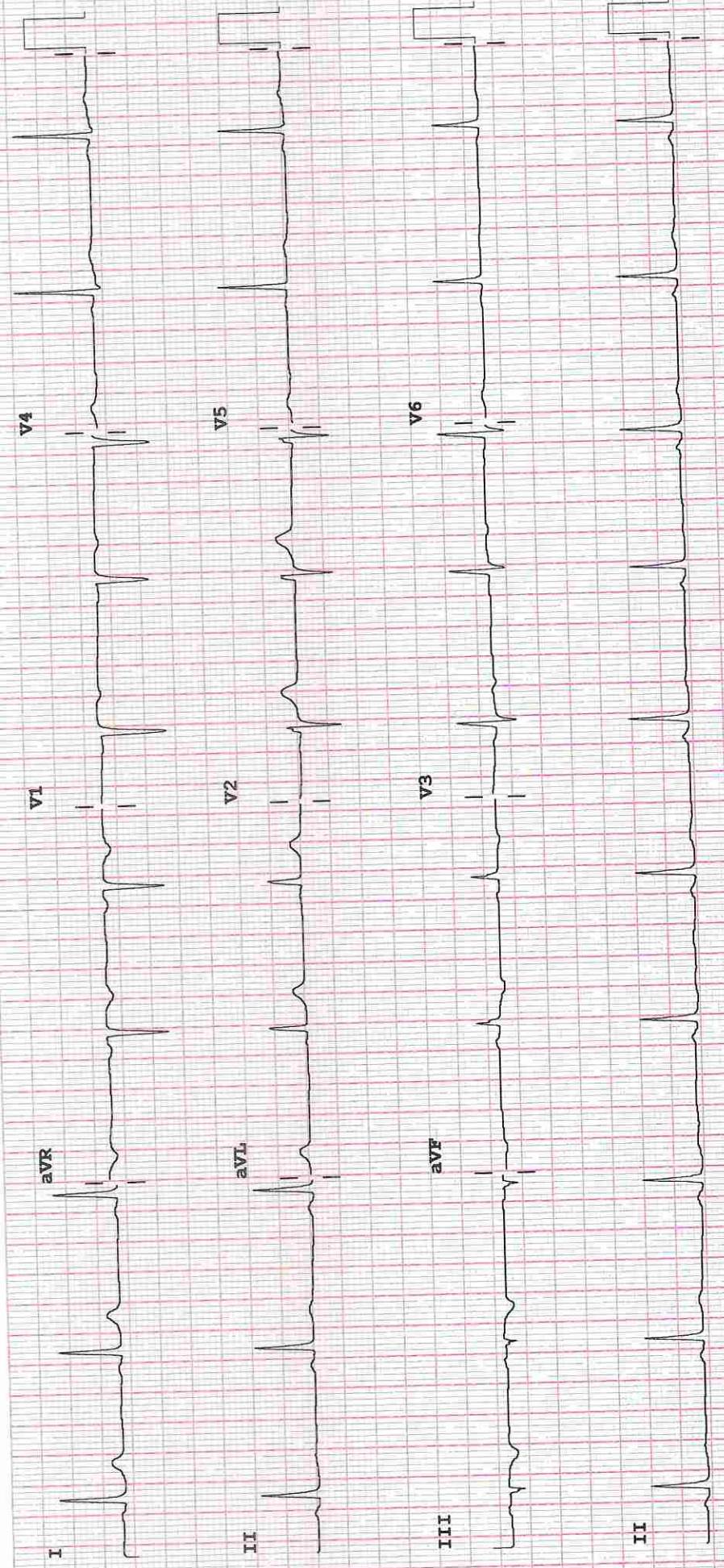
--AXIS--

P 44
QRS 22
T -19

-- BORDERLINE ECG --

Unconfirmed Diagnosis

12 Lead; Standard Placement



F 50~ 0.50-100 Hz W
100B CL P?

Speed: 25 mm/sec
Limb: 10 mm/mV
Chest: 10.0 mm/mV
Device:



DEPARTMENT OF RADIOLOGY

Date: 15/Jan/2024

Name: Mrs. Ranu Patel

Age | Sex: 41 YEAR(S) | Female

Order Station : FO-OPD

Bed Name :

UHID | Episode No : 12921812 | 2621/24/1501

Order No | Order Date: 1501/PN/OP/2401/5415 | 13-Jan-2024

Admitted On | Reporting Date : 15-Jan-2024 15:25:53

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 are unremarkable.

DR. CHETAN KHADKE
M.D. (Radiologist)



Patient Name	: Ranu Patel	Patient ID	: 12921812
Sex / Age	: F / 41Y 1M 24D	Accession No.	: PHC.7306255
Modality	: US	Scan DateTime	: 13-01-2024 13:55:58
IPID No	: 2621/24/1501	ReportDatetime	: 13-01-2024 14:57:09

USG – WHOLE ABDOMEN

LIVER is normal in size and 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 mildly enlarged in size (13.6 cm) and normal in echogenicity.

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

Right kidney measures 10.2 x 3.9 cm.

Left kidney measures 11.7 x 4.8 cm.

PANCREAS: Head and body of pancreas is visualised and appears normal. 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.

UTERUS is normal in size, measuring 8.1 x 5.9 x 3.4 cm.
Endometrium measures 7 mm in thickness.

Both ovaries are normal.

Right ovary measures 2.5 x 1.5 cm.

Left ovary measures 3.3 x 2.3 cm.

No evidence of ascites.

Impression:

- Mild splenomegaly.

DR. KUNAL NIGAM
M.D. (Radiologist)



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USG - BREAST

Findings:

Bilateral breast parenchyma appears normal.

No evidence of solid or cystic lesion.

No dilated ducts are noted.

The fibroglandular architecture is well maintained.

Retromammory soft tissues appear normal.

No evidence of axillary lymphadenopathy.

Impression:

- No significant abnormality detected.

YK

DR. YOGINI SHAH

MRD., DNB. (Radiologist)